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CHRONIC RECURRENT PANCREATITIS

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Chronic recurrent pancreatitis, also known as chronic relapsing pancreatitis, has always been a problem of some magnitude for the surgeon. In writing this article we have made an attempt to give a brief summary, from a practical point of view, of the anatomical and physiological background of the problem; to describe some of the many and varied approaches to the problem; and to formulate a new approach, which, we hope, may lead to further discussion and research.

Anatomy

The pancreas consists of a head and neck, with the uncinate process lying in front of the second lumbar vertebra, and the body and tail extending to the hilum of the spleen.

The following arteries contribute to the blood supply of the pancreas:

(*a*) The superior pancreatic artery—a branch of the coeliac axis, or the hepatic artery, or the splenic artery.

(*b*) The inferior pancreatic artery—a branch of the superior pancreatic artery or the superior mesenteric artery.

(*c*) The anterior superior branch of the pancreaticoduodenal artery.

(*d*) The posterior superior pancreaticoduodenal artery. Both (*c*) and (*d*) are branches of the gastroduodenal artery.

(*e*) The supraduodenal artery which is also a branch of the gastroduodenal artery.

The principal venous drainage of the pancreas is into the superior mesenteric vein *via* the termination of the inferior mesenteric veins; through the terminations of the left gastric vein, the splenic vein, the right gastro-epiploic vein, the anterior superior pancreaticoduodenal vein, the anterior inferior pancreaticoduodenal vein, the inferior pancreatic vein, the pancreatic cervical vein, and through several tributaries directly into the splenic vein. It is noteworthy that no vein enters the anterior surface of the portal vein. The arterial supply to, and venous drainage from, the head and neck are entirely separate from those of the body and tail, thus constituting two separate entities.

The pancreatic duct system is composed of minor tributaries which enter the main duct at a right angle. This duct drains into the ampulla of Vater, and from it an accessory duct, the duct of Santorini, enters the duodenum on its own.

There are 4 principal variations in the mode of entry of the duct into the duodenum: (1) In 29% of cases two separate channels are present—one for the pancreatic duct and one for the bile duct at a common opening, (2) in 37% both ducts have a common opening which is short, (3) in 30% both ducts have a common opening which is long, and (4) in 2% there is a single opening for the common duct and the pancreatic duct draining into the common duct.

The diameter of the orifice of the ampulla of Vater varies from 1·5 to 4·5 mm., and the average width is 3 mm. The length of the ampulla from the duodenal surface to the tip of the papilla or the summit of the septum varies from 1 to 14 mm. This fact is of importance in regard to the reflux biliary theory as a factor in the causation of pancreatitis.

The musculature of the sphincter itself consists of circular fibres passing round each duct and some passing round both ducts, and Doubilet and Mulholland[1] demonstrated a common channel in 48 out of 49 operated cases; in these cases closure of the sphincter would close both ducts.

Histology

The pancreas is divided into lobes and lobules by septa consisting of connective tissue. It is covered on its anterior aspect by the posterior parietal peritoneum, but the gland itself posesses no true capsule. Neural elements arising from the sympathetic and parasympathetic systems traverse the interlobular septa. The functional components of the pancreas consist of two types of cellular elements. One of these is concerned with external secretion, (the function of the larger portion of the pancreas) and is arranged structurally in the form of acini consisting of a layer of pyramidal or cuboidal cells surrounding a central lumen which opens into a tributary of the ductal system. The lesser portion of the pancreas is taken up with internal secretion in which the cellular elements are arranged in insular fashion and known as the islands of Langerhans, numbering from 200,000 to 2,000,000 in the human gland. These islands are scattered irregularly throughout the gland and are more numerous in the tail than elsewhere. They are composed of several types of cells mostly containing granules in the cytoplasm. The alpha cells are acidophilic, while the beta series are basophilic. There is also a group of cells in the islands that contains no granules. Islet cells are arranged in chains, separated by a generous network of capillaries. There is some speculation whether the mature acinar cell can, under adequate stimulation, differentiate into an islet cell, thus permitting the number of islet cells to vary in proportion to the demand.

Physiology

The physiology of the pancreas is manifested by two secretions—an external secretion derived from the acinar cells and an internal secretion derived from the islet cells. Dragstedt has imputed to the function of this gland the manufacture of lipocaic, a hormone concerned with fat metabolism.

Normal pancreatic juice is colourless, turbid, and alkaline, and is secreted as a response to ingested food. The normal output of pancreatic juice is 1,200 - 2,000 c.c. per 24 hours, and the enzymes contained in it are trypsin, chymotrypsin, lipase, amylase, renin and maltase. The pH is between 8·3 - 8·6, the specific gravity is 1015 and the juice contains 8 g. of protein per 100 c.c. The chloride content is between 35 - 97 mEq./l., the bicarbonate 30 - 74 mEq./l., the sodium 134 - 142 mEq./l., the potassium 4·7 - 5·4 mEq./l. and the calcium 0·4 - 4·7 mg. per 100 c.c.

The proteolytic enzyme of the pancreatic juice enters the duodenum as trypsinogen which is inert, but is activated into trypsin by enterokinase elaborated by the duodenal mucosa; it also enters as chymotrypsin. The tryptic activity is that of breaking down the peptide linkage of proteose, peptones, and higher polypeptides of the broken down protein elements that have been digested by pepsin in the stomach.

The amylolytic enzyme of the pancreas acts in a neutral medium. It is suggested that secretin is present in the duodenal mucosa in an active form. It has also been stated that secretin is present in the inert form of pro-secretin, which becomes activated by the presence of bile. Secretin is formed as a result of the stimulus of ingestion of food, and in turn stimulates the flow of bile and succus entericus.

The synthesis of protein in the pancreas is elaborate. It is not necessary for our purposes to give a detailed description of how the granules are transformed (by chemical reaction) by the action of erepsin. The lypolytic enzyme is called steapsin, which digests fats into glycerine and fatty acids. The amylase in the pancreatic juice converts carbohydrates into dextrines. The hormone secretin from the duodenal mucosa is the prime factor in promoting pancreatic secretion. Stimulation of the vagus nerve causes the flow of pancreatic juices but never that of secretin. The sympathetic fibres play only a minor role in the regulation of pancreatic secretion.

The action of various drugs on the pancreas is well known, and we do not propose to elaborate further on this, except to state that alcohol diminishes the volume of secretin. One litre of 5% glucose in water intravenously increases the flow by 50%, and cortisone has no effect on the pancreatic secretion. Antibiotics are not excreted in the pancreas, with the exception of sulphadiazine where the excretion is high, but penicillin and streptomycin are

not excreted. With regard to the internal secretion no further mention need be made of the well-known work of Banting and Best. The question of lipocaic is still under investigation, and we are not prepared at this stage to express a dogmatic view on this subject.

Aetiology

Chronic pancreatitis has been recognized for many years, but Comfort and his associates[2,3] first described it as a clinical entity in 1946. They described a syndrome characterized by attacks of upper abdominal pain with a variable degree of acinar and islet dysfunction followed by certain sequelae. It is now recognized as a separate clinical entity.

Chronic relapsing pancreatitis must be considered as a continuation or progression of acute pancreatitis, either in a mild or severe form—the recurrent attacks ultimately leading to persistent chronic symptoms and permanent physiological disturbance.

Sex. The ratio of male to female patients is 2 : 1, which rather minimizes gall-bladder disease as a causative factor.

Age. The ages of the 27 cases described by Comfort *et al.*[2] vary between 10 and 75 years. In a group of 29 cases observed in the Lahey Clinic[9] the ages vary from 20 to 64. We have had a child of 9 with recurrent pancreatitis and a man of 87 with the same disease.

Obesity, whilst common in acute pancreatitis, is not the rule in the chronic disease, since patients with the chronic disease tend to lose weight rapidly.

Alcohol. It is common to find that chronic alcoholism antedates the history of pancreatitis, and it would be fair to state that chronic alcoholism is a concomitant in between 40 - 50% of cases in this disease.

Trauma is an uncommon but nevertheless definite cause of the disease, and has been reported in 3 out of 38 cases of a series by Cattell and Warren.[4]

Disease of the biliary tract. After a detailed analysis of their clinical material Comfort *et al.*[2] concluded:

1. Inflammatory disease of the gall bladder was not a requisite of the development of chronic relapsing pancreatitis, and

2. When the pathological processes occurred simultaneously or in association, the preponderance of evidence suggested that the disease of the biliary tract is secondary to the pancreatic disease.

Common channel. Doubilet and Mulholland[5-7] more recently stated that the reflux of bile through a common pancreaticobiliary channel is primarily responsible for chronic relapsing pancreatitis. Archibald[8] popularized the theory that spasm of the sphincter of Oddi caused reflux of bile into the pancreas and in this way generated the acute disease. Doubilet and Mulholland[5] have found a common channel in most instances, and have also found that spasm of the ampulla of Vater can be induced by the application of dilute hydrochloric acid to the duodenal papilla.

Infection. There is no definite evidence that infection plays a part in the cause of this disease.

Types of Chronic Pancreatitis

There are 4 main types of chronic pancreatitis:

1. The diffuse type in which the entire gland is involved. This is often found on exploration of the abdomen.
2. Chronic pancreatitis with multilocular cystic changes.
3. Chronic pancreatitis with pancreatic lithiasis or calcinosis.
4. Localized chronic pancreatitis secondary to trauma. Pancreatic lithiasis represents the final stage in the progression of chronic relapsing pancreatitis.

The pathogenesis of pancreatic lithiasis is unknown, but the precipitating factors appear to be: (*a*) Obstruction, (*b*) stagnation, (*c*) infection (questionable), and (*d*) fibrosis and autodigestion with fat necrosis.

Pathology

Early in the disease the gland is enlarged, firm, and somewhat pale in appearance. The limits of the gland are indistinct and peripancreatic oedema is present. As the disease progresses the pancreas becomes larger and more indurated, and the body and tail lose their prismatic cross-section to become more rounded. The gland becomes fixed to surrounding structures and loses what limited mobility it had. Adjacent structures may become adherent. Pressure on the portal and superior mesenteric veins may cause portal hypertension, and the veins in the region of the stomach especially are engorged.

The head of the gland is, as a rule, disproportionately large and solitary or multiple cysts may be seen or palpated. The duct of Wirsung is frequently enlarged, and the cut surface of the gland is fibrotic and gritty, and does not bleed as readily as the normal gland.

Areas of calcification may be found, and stones varying in size from a fraction of a mm. to 3 cm. in diameter may be present; they are firm, white, round and smooth, conforming to the contour of the duct. They are laminated and range in number from 1 to 300.

SYMPTOMS AND SIGNS

Chronic pancreatitis is a difficult diagnosis to make, but if the condition is borne in mind there is no reason why an accurate diagnosis should not be established. The symptoms are as follows:

1. *Pain*

(i) Abdominal pain, which is colicky in nature, is present. As a rule the pain is severe in the acute attacks, and less severe in the recurrent attacks.

(ii) The attacks are episodic, with intervals of freedom.

(iii) With chronicity constant pain is a feature, so much so that these patients often become chronic alcoholics as a result of pain, if they are not such already, or they become morphine addicts. Of Cattell's series[4] 58% were addicted to narcotics. The pain is epigastric and is referred to the left hypochondrium and through to the back. Pylorospasm is a frequent feature. Attacks are worse at night, and the patient sits doubled up with his hands folded across his abdomen.

2. *Nausea and Vomiting*

(i) This usually appears before the onset of the pain.

(ii) Persistent vomiting is due to duodenal obstruction when it occurs.

3. *Diarrhoea, Steatorrhoea, Creatorrhoea*

These signs are found in 50% of cases, are related to the attacks and may become so severe as to produce a negative nitrogen balance.

4. *Constipation*

This is due, when it occurs to: (*a*) Diminished food intake, (*b*) reduction in fat in the bowel, and (*c*) narcotics.

5. *Weight Loss*

Weight loss is frequent, and is due to anorexia and failure of fat absorption.

6. *Jaundice*

Jaundice was observed in 18 out of 65 cases collected by Haggard and Kirtley[9] and in 26% of cases at the Lahey Clinic. Most of these cases are due to stones in the common duct, but pancreatic calcification and fibrosis is often associated with hepatocellular disease, which may account for the jaundice, or compression of the common bile duct in the pancreas.

7. *Diabetes Mellitus*

This is a late complication, and is often latent, but diabetes associated with upper abdominal pain should suggest the diagnosis of pancreatitis.

8. *Physical Signs*

Physical signs are totally absent, except for cyst formation.

9. *Laboratory Investigations*

Undue stress has been placed on these investigations, but they may be of some assistance, e.g. (*a*) serum amylase is raised only in the acute episodes; (*b*) the diminution of pancreatic secretion in response to intravenous secretin as determined by analysis of the duodenal contents (the secretin test), although revealing in some instances, is a tedious and

unnecessary procedure; (*c*) alterations in the glucose tolerance occur in approximately 1/3rd of the cases; and (*d*) fat and meat fibres are found in the stool. This is a most inconstant finding.

10. *X-ray Findings*

Pancreatic calculi may be demonstrated radiologically, but the principal features are distortions or displacements of adjacent viscera. The duodenal C is widened if the head is enlarged, or large cysts cause pressure deformities of the stomach. The most helpful sign is the presence of calcification in the pancreas, and it must be remembered in the radiological technique to scrutinize the control film carefully for a solitary stone in the region of the ampulla.

COMPLICATIONS

The following complications occur in chronic pancreatitis: (1) Diabetes mellitus, (2) pancreatic cysts, (3) pancreatic abscesses, (4) pancreatic haemorrhage, (5) portal hypertension, (6) biliary cirrhosis, and (7) carcinoma of the pancreas, which supervenes in a small percentage of cases.

TREATMENT

The wide variety of therapeutic measures at present advocated for this disease is an indication of the inadequacy of th treatment of the condition. The programme that has been adopted can be summarized as follows:

1. The Elimination of Causative Factors

The elimination of all possible causative factors, such as alcoholism, biliary-tract disease, diabetic errors, etc.

2. A Medical Regime

A careful medical regime, consisting of: (i) A bland diet free of fruit and vegetables, with a reduction, but no elimination, of fats; (ii) the prohibition of alcohol; (iii) the elimination of morphia, which is a cause of spasm of the sphincter of Oddi; and (iv) the administration of 6-8 g. of pancreatin per day, of insulin where necessary, of bile salts and of dehydrocholine, 1 tablet 3 times daily before meals.

3. Surgical Manoeuvres

Despite careful medical treatment, the majority of cases will require surgery, and the surgical manoeuvres can be listed as follows:

1. *Indirect methods of treatment*

These include:

(*a*) *Biliary tract procedures*, i.e. (1) Cholecystectomy-choledochostomy, (2) biliary-intestinal anastomosis, and (3) sphincterotomy.

(*b*) *Gastro-intestinal diversion*, i.e. (1) gastro-enterostomy, (2) pyloric exclusion, and (3) gastrectomy.

(*c*) *Nerve interruption*, i.e. (1) Sympathectomy (thoracolumbar and splanchnicectomy) and (2) vagotomy.

2. *Direct methods of treatment*

These include: (*a*) Drainage of cysts, (*b*) lithotomy, (*c*) anastomosis (continuity and diversion), and (*d*) resection (distal pancreatectomy, pancreatoduodenectomy, and total pancreatectomy).

Comment

Cholecystectomy and choledochostomy are only indicated in the presence of biliary disease, and this, as stated earlier, is not a frequent accompaniment of pancreatitis. Biliary intestinal anastomosis may be necessary as a result of the pancreatic disease. It is, however, not of much value in the treatment of the disease.

Sphincterotomy is advised by Doubilet and Mulholland;[5] it is also the procedure adopted by Rodney Smith.[10] These authors report good results, but in our experience only 40% of patients obtain relief by this procedure, which should always be carried out by the transduodenal route. Cattell[11] inserts the long limb of the T-tube into the duodenum, but we have had 2 cases where this produced acute pancreatitis.

Gastro-enterostomy is only indicated in obstruction. This procedure has been considered of value in some quarters because of the diversion of the food and gastric contents. Gastrectomy has been advocated in order to obtain a similar result, but we do not believe that this procedure should be attempted except where there is existing disease of the stomach or duodenum.

Sympathectomy and splanchnicectomy have been advised for relief of pain, and some favourable results have been reported in employing these procedures, but recourse to them should be the last resort since they are only symptomatic methods of treatment.

Vagotomy, in our opinion, is not a worthwhile undertaking. Cysts will require either internal or external drainage. Cattell[11] is still of the opinion that external drainage is the most satisfactory procedure, but we have found that internal drainage, preferably to the stomach, has met with success.

Pancreatic lithotomy is indicated where stones are observed. Some workers have advised ligations of the pancreatic ducts to produce atrophy of the acinar portion of the pancreas, but if this is successful it will produce a chronic state of nutritional deficiency.

Anastomosis of the duct of Wirsung has been advised by Cattell.[12] This procedure does not appear to have any appreciable applicability for the duodenal end of the duct, but is valuable for the distal end where there is obstruction anywhere between the neck and the tail; the procedure is then accomplished by distal pancreatectomy with anastomosis of the remaining portion of the pancreas to the jejunostomy by the Roux-en-Y technique.

Pancreatoduodenectomy may be indicated on rare occasions —usually in association with carcinoma or multiple calculi.

The same applies to total pancreatectomy, but the control of diabetes after this operation is difficult, because of the patient's extreme sensitivity to insulin.

RECENT WORK

Having reviewed the position in regard to pancreatitis up to the date when we undertook the project which we are about to describe, we will now deal with the recent work in relation to this disease. This project was commenced in 1951 when we began doing cholangiography. We were impressed with the fact that on many occasions the pancreatic duct was visualized during this procedure. We felt that it was reasonable that if the pancreatic duct could be totally visualized there might be a better understanding of the depressing disease of chronic recurrent pancreatitis.

The first problem was that we had no established normal. At that time (1951) no such procedure had been undertaken and operative pancreatography was totally unknown. The problem was how to approach the pancreatic duct, and it was only with the advent of sphincterotomy in 1953 that we realized that this was the mode of access.

Cases were few and far between and, in 1956 when only 3 cases had been dealt with, an article was published by Doubilet and Mulholland[13-14] describing a project similar to

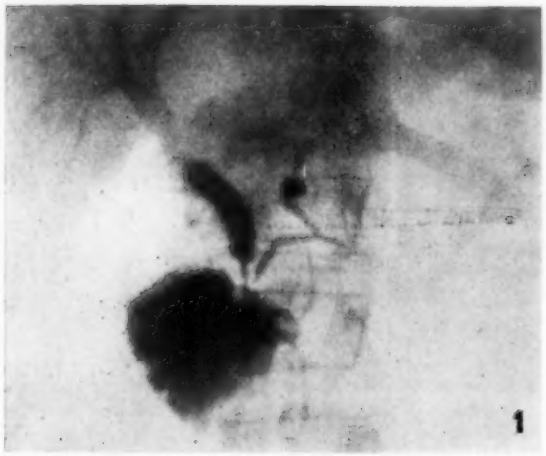

Fig. 1. Visualization of the pancreatic ducts during operative cholangiography.

ours. Within a short while they published an evaluation of this work in March 1957.[15]

Our theory was that chronic relapsing pancreatitis was due, in the main, to causes within the pancreatic duct system, and all our investigations were related to this factor.

We felt that the following information could be obtained by pancreatography:

1. Obstruction at the entrance of the duct at the sphincter of Oddi.
2. Stricture of the pancreatic duct anywhere in its course.
3. The discovery and accurate pin-pointing of pancreatic calculi.
4. The demonstration of ductal ectasia.
5. Cyst formation, either large, small, single, or multiple.
6. The presence of an abnormal duct system.

It was also hoped that this procedure would be of value in demonstrating small tumours of the pancreas, and in demonstrating extrinsic pressure on the pancreatic duct system from outside sources. Furthermore, it was felt that the operation of distal pancreatico-jejunostomy or partial resection of the pancreas, as first described by Aird and Buckwalter[16] in 1955, would be indicated after the demonstration of pathology in the duct system as shown in pancreatography.

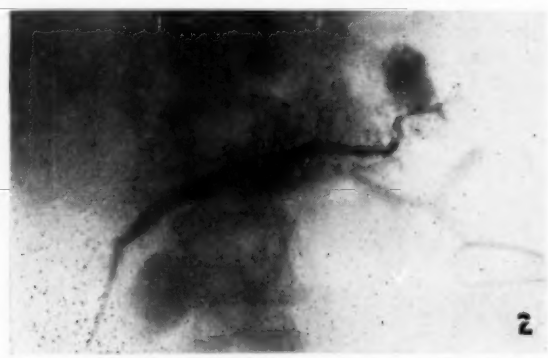

Fig. 2. Operative pancreatogram showing a grossly dilated duct with a terminal cyst.

TECHNIQUE

The abdomen is opened by a Kocher's incision. The gall-bladder and biliary tract are explored, and a cholangiogram is performed on the table. If any disease of the biliary tree is found it is dealt with as required, and the common duct is in any event opened. Thereafter the duodenum is opened and a sphincterotomy is performed, whereby only the musculature of the sphincter is cut through at 12 o'clock, and the musculature of the duodenum is not disturbed.

At this stage the opening of the main pancreatic duct can usually be visualized in its position at 5 o'clock opening at the sphincter of Oddi. If the duct opening is not found in this position, it usually opens ½ inch above this into the common duct itself, and can be demonstrated in that position. The duct having been opened, a fine ureteric catheter is passed into it, and 3 c.c. of urografin 60% is introduced into the catheter. X-ray pictures are then taken. It is important at this stage to realize, as we have found from experience, that the maximum capacity of the pancreatic duct system is never more than 3 c.c. If any resistance is therefore met with when the injection is performed, it should be stopped immediately, otherwise the duct will be blown—usually at the terminal end, with the possible establishment of a localized area of pancreatitis.

If a stricture is discovered in the proximal portion of the duct, it is dilated with ureteric dilators, and at the completion of the dilatation a further pancreatogram is done. If the stricture is found in the distal end of the duct, then this together with the distal portion of the gland is resected. If the stricture is more towards the head and neck, then the distal portion of the duct is anastomosed to the jejunum by the Roux-en-Y technique. If calculi are discovered they are dealt with either by removal or by resection, and similarly, any local pathology in the gland itself can be handled according to what is discovered on X-ray.

If no pathology is discovered then the sphincterotomy has acted as a therapeutic procedure for the treatment of the pancreatitis.

The following case reports are of interest in this connection:

Case 1

Mr. L., aged 48, was seen on 17 April 1957. His history was that during the war, whilst on active service, he had had several attacks of pain in the abdomen, and an ulcer was diagnosed. He was X-rayed and no ulcer was found, but he was subsequently discharged from the army with the diagnosis of 'gastric trouble'. On the night that we were asked to see him he was a desperately ill man, with a history that 48 hours before this date he had an attack of pain in the left loin and the back, radiating across the upper abdomen. He had vomited incessantly for 36 hours, the pain was constant, and had become progressively worse. His blood pressure was 90/60 mm. Hg, his temperature was 100°F, his pulse rate was 68, his upper abdomen was distended, and the whole abdomen was completely rigid and silent. A diagnosis of acute pancreatitis was made, and he was admitted to the nursing home. At that stage, 48 hours after the commencement of his illness, his serum amylase was 640 Somogyi units, and his white-blood count was 21,000 per c.mm. He was treated conservatively with intravenous fluids and antibiotics, and he gradually improved and was discharged from the nursing home 9 days later. Sixteen days after his discharge from the nursing home he had a recurrence of the same type of pain, and was readmitted to the nursing home. At this stage his serum amylase was 1,600 Somogyi units, and his white-blood count was 9,000 per c.mm. He was treated conservatively again and was discharged from the nursing home approximately 10 days later. At this stage he was not entirely free of symptoms and, because of the persistence of his pain, he was readmitted a week later and operated on. The pancreas was large, swollen

and oedematous. The common bile duct was opened, and no pathology was found. The gall bladder appeared to be entirely normal. The duodenum was opened and a sphincterotomy was performed. The pancreatic duct opening was visualized, and a ureteric catheter inserted and the dye injected. At a distance of 1½ inches from the opening a stricture was found. This was dilated and the patient was re-X-rayed, when it was found that the pancreatic duct was now normal. His progress thereafter was satisfactory, and within 14 days he was discharged from the nursing home. Subsequent follow-up reveals that this patient, almost 2 years later, has had no further trouble, and is doing a full normal day's work on a normal full diet.

Case 2

Mr. M., aged 36, was admitted to a urological ward with a history that he had been on an alcoholic spree for approximately a week before the date of admission on 30 May 1958. On the day before admission he had developed acute colicky pain in both loins, which radiated round to the front, especially to the left, where it reached the left iliac fossa. The registrar, an acute observer, decided that this case was possibly one of acute pancreatitis, and the serum amylase was 458 Street-Close units. This patient was treated conservatively without his symptoms completely resolving, and was operated upon on 8 July 1958 through a Kocher's incision. The pancreas, once again, was thick and oedematous in its entire extent. The gall bladder was normal, and the common duct was explored and also found to be normal. The duodenum was opened and a sphincterotomy was performed. A ureteric catheter was introduced into the duct of Wirsung, and a stricture was found at its opening. This was dilated up eventually to a No. 2 De Bakey, and after the dilatation there was a gush of pancreatic juice into the operative field. The patient made an uninterrupted recovery, and was discharged from hospital 22 days later. He has been followed up since, and on only one occasion did he have a mild attack of lower abdominal pain, when he was readmitted to hospital and a diagnosis of diverticulitis was established. As far as his pancreas is concerned, he has had no trouble for the past 10 months.

Case 3

Mr. I., aged 33, is a confirmed alcoholic. He had suffered from abdominal pain on and off for some years. He had been readmitted to hospital on several occasions with attacks of pancreatitis.

He was dealt with in the same way as the other two cases, and he was found to have a stricture in the middle of the pancreatic duct. This case was dealt with by an anastomosis of the distal portion of his pancreas to the jejunum by the Roux-en-Y technique.

On 15 October 1957 there was a complete resolution of symptoms, and he was discharged from hospital 11 days later. He has been readmitted on two occasions, once in June 1958 and again on 16 July 1958, on both occasions having had an acute alcoholic episode with a mild recurrence of symptoms. Despite psychiatric therapy he still continues to remain a chronic alcoholic, and we are afraid that the result will not be good in his case if this state of affairs persists.

Case 4

Mr. J. was admitted to hospital on 7 June 1958, having had a previous attack of pancreatitis. This admission was for a very acute episode, which was treated conservatively and subsided within a few days, but kept on recurring. During an exploratory examination on 24 June 1958 he was dealt with in a similar manner to the cases mentioned above. In this case ectopic pancreatic tissue was found in the duodenum in the region of the ampulla, which was markedly engorged and oedematous. The sphincter was only found with difficulty, and a sphincterotomy was performed. The pancreatic duct was dilated, but the pancreatogram was unsatisfactory because of a technical radiological difficulty.

He was returned to the ward, but developed a massive collapse of both lungs the same night. He died 3 days later. Autopsy revealed an extensive collapse of the lungs with bronchopneumonia and a complete fatty infiltration of the liver with a severe degree of intra-acinar fibrosis. Sections of the kidney showed tubular necrosis. The pancreas was still oedematous and showed evidence of chronic pancreatitis, and the duct itself was thickened and fibrosed and small foci of epithelium were embedded in the duct.

Whilst the literature on this subject is still very small, nevertheless, Pollock[17] in a recent article reported on his experiences in doing 33 pancreatograms on cadavers and 11 on live patients, and he is prepared to condemn this operation. Other writers to date feel as we do that there is a future for this procedure.

CONCLUSION

It is too early at this stage to evaluate results, and the number of cases, viz. 8, is still too small, but we are satisfied that this procedure yields valuable information, and has added fundamental knowledge to a subject about which we are very ignorant. Its use, we are sure, will throw light on what was previously a valley of darkness.

SUMMARY

1. A brief summary is given, from a practical point of view, of the anatomical and physiological background of the problem of chronic recurrent pancreatitis.

2. A summary is given of the wide variety of therapeutic measures at present advocated for this disease.

3. The technique of operative cholangiography and pancreatography is described and 4 case studies are quoted.

4. It is concluded that the procedure described has added fundamental knowledge to a subject about which little definite information is available.

REFERENCES

1. Doubilet, H. and Mulholland, J. H. (1948): Surg. Gynec. Obstet., **86**, 295.
2. Comfort, M. W., Gambill, E. E. and Baggenstoss, A. H. (1946): Gastroenterology, **6**, 239.
3. *Idem* (1946): *Ibid.*, **6**, 376.
4. Cattell, R. B. and Warren, K. W. (1953): *Surgery of the Pancreas.* Philadelphia and London: W. B. Saunders.
5. Doubilet, H. and Mulholland, J. H. (1948): Ann. Surg., **128**, 609.
6. *Idem* (1949): Surg. Clin. N. Amer., **29**, 339.
7. *Idem* (1949): N.Y. St. J. Med., **49**, 2938.
8. Archibald, E. (1929): Ann. Surg., **90**, 809.
9. Haggard, W. D. and Kirtley, J. A. Jr. (1939): *Ibid.*, **109**, 809.
10. Smith, R. Personal discussion.
11. Cattell, R. B. (1948): Surg. Clin. N. Amer., **28**, 659.
12. *Idem* (1947): *Ibid.*, **27**, 636.
13. Doubilet, H. and Mulholland, J. H. (1956): *Ibid.*, **36**, 385.
14. *Idem* (1956): J. Amer. Med. Assoc., **160**, 521.
15. Doubilet H., Poppel. M. H. and Mulholland, J. H. (1957): *Ibid.*, **163**, 1027.
16. Aird, I. and Buckwalter, J. A. (1955): Brit. J. Surg., **42**, 491.
17. Pollock, A. V. (1958): Surg. Gynec. Obstet., **107**, 765.

BOEKE ONTVANG : BOOKS RECEIVED

Water Supply for Rural Areas and Small Communities. By E. G. Wagner and J. N. Lanoix. Monograph Series, No. 42. Pp. 340. 85 figures. £1 5s. Local Sales Agents: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724, Pretoria. Geneva: World Health Organization.

Immunological and Haematological Surveys. Report of a study Group. Technical Report Series No. 181. Pp. 35. 1s. 9d. Also published in French and Spanish. Local Sales Agent: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724, Pretoria. Geneva: World Health Organization. 1959.

European Technical Conference on Food-borne Infections and Intoxications. Report. Technical Report Series No. 184. Pp. 18. 1s. 9d. Also published in French and Spanish. Local Sales Agent: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724, Pretoria. Geneva: World Health Organization. 1959.

The Student and Mental Health. Edited by D. H. Funkenstein, M.D. Pp. xxv + 495. 35s. net. London: H. K. Lewis & Co. Ltd. 1959.

The Diabetic Life—Its Control by Diet and Insulin and Oral Treatment by Sulphonyl-ureas. 16th edition. By R. D. Lawrence, M.A., M.D., F.R.C.P. (Lond.). Pp. x + 230. 19 illustrations. 15s. net. London: J. & A. Churchill Ltd. 1960.

A Way of Life and Selected Writings of Sir William Osler. 12 *July 1849 to 29 December 1919.* (Formerly titled *Selected Writings of Sir William Osler.*) Edited by a Committee of the Osler Club of London with the help of W. W. Francis, *et al.* Pp. xx+278+16. 5 illustrations. $1.50. New York: Dover Publications, Inc. 1959.

Hypnosis, Fact and Fiction. By F. L. Marcuse. Pp. 224. 3s. 6d. Middlesex: Penguin Books. 1959.

SENSITIVITY REACTION TO POLIOMYELITIS VACCINE

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Poliomyelitis vaccine is exceedingly safe and very rarely causes any unpleasant sequelae. This has been the experience in the United States of America, the United Kingdom and South Africa.

The following case report illustrates an anaphylactic reaction following 4 hours after the injection of poliomyelitis vaccine. An unusual feature was the development of tetany.

CASE REPORT

A 26-year-old European housewife has been suffering from hay fever since the age of 14 years. For the past 18 months she had attacks which were considered to be asthmatic. A skin test was carried out by a specialist in allergy and the patient was found to be sensitive to mixed grass pollen. Desensitization was carried out. During the course of the desensitization, after the 5th injection, she required to have poliomyelitis vaccine. The first injection of vaccine (prepared by the Poliomyelitis Research Foundation, Johannesburg) caused no difficulty. A month later the second injection was given; within 4 hours she developed severe abdominal cramps and vomiting. At first there was no respiratory embarrassment, but when the doctor was called, the patient was collapsed and there was evidence of peripheral circulatory failure and peripheral cyanosis. The pulse was fast, blood pressure unrecordable, respiration was rapid and wheezing, and she complained of severe abdominal pain. Manifestations of tetany were observed, the patient exhibiting spontaneous carpopedal spasm and irritability of the facial nerve (Chvostek's sign). Adrenaline was administered immediately (0·5 ml. of a 1 in 1,000 solution, subcutaneously) followed by 15 mg. of mephentermine sulphate (wyamine) intravenously. There was an improvement for the next half an hour, but then the patient's condition deteriorated. Promethazine (phenergan) was given intramuscularly with good effect. Calcium gluconate solution intravenously appeared to improve the manifestations of tetany though irritability of the facial nerve persisted for several hours after the general condition had improved.

Treatment with antihistamine drugs and intravenous calcium gluconate was continued for 24 hours. She was then quite well and no other manifestations of sensitivity developed. The patient had received several injections of penicillin 5 years and 7 years previously, with no ill effects.

COMMENT

Injections of poliomyelitis vaccine have been reported to cause skin rashes as well as anaphylactic reactions. The vaccine generally contains culture medium, horse serum or bovine serum, soluble monkey protein from blood or kidney, formaldehyde, preservatives, polio virus, and antibiotics (penicillin). The cultures are well washed to get rid of the serum before the addition of the virus phase of the medium and, according to regulations, the dilution of serum should be less than 1 in 1,000,000.[1]

It is generally assumed that the penicillin content, however minute, acts as the allergen in reactions to polio vaccine. This assumption is based upon the observation that a penicillin-free vaccine does not cause reactions in sensitive patients and upon reports that penicillinase protects against reactions. Zimmerman[2] described 6 patients, all with a previous history of sensitivity to penicillin, who developed urticaria or bullous dermatitis a few days after poliomyelitis vaccination. In all these patients the reaction to the vaccine duplicated the previous reaction to penicillin and there was rapid clearing of the condition after a single injection of penicillinase. The patients did not react when a penicillin-free vaccine was administered subsequently.

In the vaccine prepared by the Poliomyelitis Research Foundation, Johannesburg, the amount of penicillin added to the fluid used in preparing the cultures is 100 units per ml., that is, per one dose, but in the final vaccine the penicillin has deteriorated to such an extent that its presence can no longer be detected. However, as Gear[1] points out, though the bactericidal and static activities may have diminished to vanishing point, its allergenic capacity may not have altered to the same extent!

It has been reported from the USA[3] that vaccines prepared by commercial drug manufacturers in America contained amounts of penicillin varying from 0.001 μ./ml. to less than 20 μ./ml., but the amounts of penicillin reported to have caused anaphylactic reactions is sometimes minute. Brierlein[3] reported a patient developing shock from an intracutaneous skin test with 3 one-millionths of 1 unit of penicillin. This patient had a passive transfer skin test positive to 1 in 25 dilution of poliomyelitis vaccine.

Calculating from vaccine issues in the Union of South Africa it is believed that approximately 750,000 persons have been vaccinated and the total number of reactions reported has been small.[1] However, most of the injections have been given to children, and children rarely manifest anaphylaxis.[4] Skin rashes, usually urticaria, have been reported to the Poliomyelitis Research Foundation and in a few children suffering from asthma the inoculation has apparently precipitated attacks.

The patient described in the present paper was a highly allergic subject who had received injections of penicillin previously with no ill effect; indeed she did not react to the first dose of vaccine. It is presumed that penicillin is the offending constituent in this instance.

Of interest was the occurrence of tetany. Anaphylaxis is usually accompanied by bradypnoea, but the increased expiratory effort can conceivably cause respiratory alkalosis with resulting increase in excitability of nerve tissue. Anxiety would contribute to the process. In our patient, it was debated whether hyperventilation alone accounted for the clinical picture, but this did not explain the shock, the bronchospasm, the fall in blood pressure, the abdominal manifestations, and the response to adrenaline.

REFERENCES

1. Gear, J. H. S. (1960): Personal communication.
2. Zimmerman, M. C. (1958): J. Amer. Med. Assoc., **167**, 1807.
3. Brierlein, K. J. (1956): Ann. Allergy, **14**, 35.
4. Collins-Williams, C. and Vincent, J. (1953): *Ibid.*, **11**, 454.

VAN DIE REDAKSIE : EDITORIAL

OSTEOMIËLITIS

Dit sal ongetwyfeld 'n verrassing wees vir baie persone dat die hoogste insidensie van poliomiëlitis in die wêreld gerapporteer is uit die Rooikruis Oorlogsgedenkhospitaal vir Kinders en die Groote Schuur-hospitaal in Kaapstad.[1]

Dit is interessant om daarop te let dat dié siekte skynbaar 'n voorkeur toon vir ligter velle aangesien dit betreklik min voorkom by Bantoes ten spyte van hul relatiewe laer lewensstandaard. Waarom die minder gegoede Kleurlinge betrokke is, is nog nie verklaar nie. Die teorie dat antibiotiese middels makliker en vroeër in die verloop van die siekte beskikbaar is vir welgestelde pasiënte, word nie gestaaf deur die feit dat selfs voor 1939 die arm klasse meer bepaald aangetas was nie.

Sedert die ontdekking van penisillien het daar 'n uitgerekte tweestryd geheers tussen die voorstanders van die konservatiewe (antibiotiese) terapie en diegene wat meen dat chirurgiese dreinering saam met antibiotiese middels 'n beter metode van behandeling is. Die reeks wat onlangs in die *Tydskrif* bespreek is,[1] dui ondubbelsinnig aan dat die meer radikale vorm van behandeling beter resultate oplewer in bewese gevalle van osteomiëlitis.

Die gevalle uit Kaapstad is egter dikwels op 'n laat stadium eers gesien in pasiënte wat (subklinies) ondervoed was. Dit is meer as waarskynlik dat dit een uiterste vorm van die siekte is waarmee ons hier te doen het en dat ligter gevalle wat opklaar sonder enige antibiotiese terapie wel voorkom. Sulke gevalle kan egter nooit bewys word nie, selfs nie in retrospek nie, sodat aansprake vir konservatiewe behandeling versigtig oorweeg moet word.

Die diagnose is dikwels baie moeilik, veral in kinders met osteomiëlitis van die nek van die femur waar lokaliserende tekens soms laat voorkom. Die enigste simptome mag mankheid wees of pyn in die heup. Aan die ander uiterste is daar pasiënte wat ernstig siek is aan septisemie, wat 'n koors het van hoër as 103°F, en wat selfs in 'n toestand van delier of koma is sonder dat daar enigiets is om aan te dui dat daar etter in die nek van die femur aanwesig is. Tensy hierdie toestand in die gedagte gehou word en kragdadige behandeling ingestel word, sal die siekte voortgaan en ook die heupgewrig aantas met uiteindelike vernietiging van die kop van die femur en met tragiese verlammende gevolge. Lokale teerheid in die femorale driehoek, voor, en tussen die sitbeenknobbel en die groot trokanter van die femur, agter, sowel as 'n effense vermindering van die omtrek van die aangetaste bo-dybeen, is waardevolle kliniese tekens. Waar twyfel nog bestaan, moet ondersoek ingestel word. As die resultate negatief is sal geen skade gedoen wees nie.

Dit is goed om te onthou dat daar gedurende die afgelope drie jaar vyf kinders dood is aan osteomiëlitis in die Rooikruis Oorlogsgedenkhospitaal vir Kinders — 'n ernstige waarskuwing om nie die erns van die siekte te verontagsaam nie. Die kinders wat dood is, is almal met groot dosisse van antibiotiese middels behandel, trouens, in die geval van drie van hulle was die betrokke organisme selfs sensitief vir penisillien.

Dit is moontlik dat die kwaadaardigheid van die organisme wat die infeksie veroorsaak, in die meeste gevalle 'n stafilokokkus aureus, op die een of ander manier aan die verander is sodat die gedrag daarvan *in vitro* nie 'n ware aanduiding is van die potensiële gevaar vir die pasiënt nie, veral nie wanneer onvoldoende dosisse van antibiotiese middels gegee word nie. Dit wil voorkom of dit verstandig is om baie groot dosisse — selfs maksimum dosisse — van antibiotiese middels te gee.

Die siekte was nie net meer noodlottig in die jongste aantal jare nie, maar die jaarlikse voorkoms daarvan is ook aan die vermeerder. Meer en meer gevalle van veelvuldige aantasting van been kom voor en die moontlike teenwoordigheid van fokusse van infeksie van been wat deur uitsaaiing ontstaan het, moet gedurig in gedagte gehou word, veral as die pasiënt nie verbetering toon na behandeling nie. Daar moet by herhaling en deeglik na hierdie fokusse gesoek word en dit mag noodsaaklik wees om pleisters te verwyder om beter in staat te wees om die ledemate goed te ondersoek.

Tot onlangs was daar geen sekerheid oor hoe lank die antibiotiese behandeling moet duur nie. Verskillende outoriteite lê willekeurige maatstawwe neer wat betref 'n minimum periode, op die basis van sulke faktore soos die pasiënt se algemene toestand, temperatuur, kliniese en röntgenologiese ondersoek van die plaaslike letsel, ens. In gevalle van osteomiëlitis is hierdie faktore gewoonlik binne twee weke weer normaal, en tog sal niemand dit durf waag om die toediening van antibiotiese middels na so 'n kort tydperk te staak nie.

Die Kaapstadse ondersoekers verdien spesiale vermelding omdat hulle aangetoon het hoe belangrik dit is om die sedimentasiespoed van eritrosiete as 'n maatstaf te neem vir die duur van antibiotiese behandeling. Eers wanneer twee opeenvolgende bepalings normaal is, kan die toediening van antibiotiese middels en volledige immobilisasie gestaak word; om dit voor hierdie tyd te doen is om 'n heroplewing van die infeksie uit te lok met die sterk moontlikheid van 'n chroniese verloop daarna — die voorkoming waarvan die doel van hedendaagse behandeling is.

1. Shandling, B. (1960): S. Afr. T. Geneesk., **34**, 520.

OSTEOMYELITIS

To many it will doubtless come as a surprise that the highest published incidence of osteomyelitis in the world has been reported from the Red Cross War Memorial Children's Hospital and the Groote Schuur Hospital in Cape Town.[1] It is an interesting observation that the disease appears to have a predilection for paler skins—there is a low incidence among the Bantu in spite of their relatively lower standard of living. Why the poorer Coloured people are affected

has not been explained; the theory that antibiotics are available more readily and earlier in the course of the disease to the wealthier patient is not borne out by the fact that even before 1939 the poor were disproportionately singled out.

Since the discovery of penicillin a protracted controversy has raged between the proponents of the conservative (antibiotic) therapy and those who maintain that surgical drainage combined with antibiotics is the better treatment. The series recently reviewed in this *Journal*[1] unequivocally indicates that the more radical form of treatment yields better results in proved cases of osteomyelitis.

The Cape Town cases, however, were often seen at a late stage in patients who were (subclinically) undernourished. It is more than likely that this is one extreme of the disease and that milder cases do occur which clear up without a trace of antibiotic therapy. However, such cases can never be proved, even in retrospect, so that claims for conservative treatment must be guarded.

The diagnosis is sometimes very difficult, especially in children with osteomyelitis of the neck of the femur, where localizing signs may not appear early. The only symptoms may be a limp or pain in the hip. At the other extreme there are patients who are gravely ill with septicaemia, who have a temperature well above 103°F, and are even delirious or comatose with nothing to indicate the presence of pus in the neck of the femur. Unless this condition is borne in mind and vigorous treatment instituted, the disease will progress to involve the hip joint in the suppurative process, with ultimate destruction of the femoral head and with tragically crippling results. Local tenderness, anteriorly, in the femoral triangle, and posteriorly, between the ischial tuberosity and the greater trochanter of the femur, as well as a slight increase in girth of the affected upper thigh, are valuable clinical signs. Finally, where doubt exists, an exploration is mandatory; if it is negative, no harm will have been done.

It is as well to remember that within the last three years five children have died of osteomyelitis at the Red Cross War Memorial Children's Hospital—a sombre note of warning not to disregard the seriousness of the disease. The children who died were all treated with massive doses of antibiotics and indeed, in three of them, the causative organism was sensitive to penicillin. It is possible that the virulence of the infecting organism, in most cases a staphylococcus aureus, is changing in some way, so that its behaviour *in vitro* is not a true index of its potential danger to the patient, especially when inadequate doses are given. It would appear to be a wise policy to administer antibiotics in maximum, if not massive, doses.

Not only was the disease more fatal in recent years, but its annual incidence is on the increase. More and more cases of multiple bone involvement are being encountered, and the possible presence of metastatic osseous foci of infection should constantly be borne in mind, especially when the patient fails to respond to the treatment. These foci must diligently and repeatedly be sought, and may require removal of plasters in order to examine the limbs more thoroughly.

The duration of the antibiotic therapy has, until recently, been largely a matter of conjecture, with various authorities laying down quite arbitrary minimum periods on the basis of such factors as the patient's general condition, temperature, clinical and radiological examination of the local lesion, etc. In osteomyelitis these factors are almost all normal within a fortnight, but, surely, nobody would advise discontinuing antibiotics after a brief course of two weeks?

To Cape Town must go the credit for pointing out the valuable place of the erythrocyte sedimentation rate as a guide to the duration of antibiotic therapy. It is not until two successive estimations are normal that the antibiotics and the complete immobilization are discontinued. To do so before this time is to invite the danger of a recrudescence of the infection, with a strong likelihood of subsequent chronicity, the avoidance of which is the aim of present-day treatment.

1. Shandling, B. (1960): S. Afr. Med. J. 34, 520.

OESOPHAGEAL HIATUS HERNIA : A STUDY OF 94 CASES TREATED SURGICALLY

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Numerous operations have been described for the treatment of oesophageal hiatus hernia. Although in the hands of the authors of these techniques the procedures have not been reported as associated with an important recurrence rate, others after detailed follow-up examination have reported a discouraging number of failures. Several authors in fact, have advised that attempts at definitive repair are not worth while and that it is preferable to enlarge, rather than diminish, the hernial orifice, so that the stomach is not constricted. A similar defeatist attitude is shown by those who recommend interruption of the phrenic nerve as the solitary surgical treatment.

Medical measures only palliate the chronic state of semi-invalidism from which these patients suffer. Only surgery can restore the normal hiatal anatomy and function and so prevent the disastrous effects of reflux oesophagitis. What is needed is a standard curative operation which not only corrects the anatomical deformity but restores the normal physiology of the cardia.

In sliding hiatus hernia, the anatomical deformity is the result of 3 basic structural defects, as follows:

(1) Atrophy with a stretching and distortion of the muscular

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collar of the hiatus, so that the hiatal opening which normally fits snugly around the oesophagus becomes widened and enlarged.

(2) Disturbance of the factors that maintain the cardia in its normal position below the diaphragm. The most important of these is the phreno-oesophageal ligament, which originates in the fibrous tissue on the underside of the diaphragm, spans the hiatus, and is attached to the entire circumference of the oesophagus about 2 - 3 cm. above the gastric junction. This structure, which normally secures the cardia below the diaphragm, becomes lax and stretched and can no longer function as an anchoring mechanism.

(3) A redundant peritoneal sac, which extends through the hiatus and is located on the antero-lateral aspects of the upper part of the stomach and terminal oesophagus. Its wall is made up of peritoneum and stretched phreno-oesophageal ligament.

In para-oesophageal hernias there are only 2 structural defects. The cardia remains anchored below the diaphragm by a functionally intact phreno-oesophageal ligament. The herniation takes place through a widened hiatal ring into a sac alongside the cardia. With this type of hernia, mere closure of the widened hiatus after repositioning of the hernia will give good results. As it is the fundus of the stomach which usually herniates through the hiatus in these cases, fixation of the greater curvature to the costal arch, as advocated by Nissen,[16-19] or to the diaphragm, may help to prevent recurrence. Excision of the sac, if large, provides an extra safeguard.

With sliding hernia, however, the ideal repair involves correction of all 3 structural defects. The normal anatomy has to be restored, not only by narrowing of the hiatus and removal of the redundant sac, but by reinstatement of the factors securing the cardia below the diaphragm. This has been attempted both by the transthoracic and the trans-abdominal routes.

Many surgeons prefer to approach the hernia through the chest. Undoubtedly this approach gives rapid exposure of the hiatus. In sliding hernias, however, it is not sufficient to rely only upon the narrowing of the hiatus, as many authors have found. Thus Gertz *et al.*[11], in a series of sliding hernias treated by careful narrowing of the hiatus, found that not more than 25% of the patients could be called completely cured and no less than 50% showed recurrence on X-ray re-examination.

There are many reasons for this failure. The problem is to repair the hernia through a flat sheet of muscle, at the same time creating a reasonably elastic opening through which the oesophagus and vagus nerves can pass. The hiatus must not be closed too tightly about the oesophagus or the patient will have dysphagia. Often the crural fibres have to be closed with some tension, so that one gets the impression that the sutures may eventually cut through the soft muscular tissue and so defeat the purpose for which they were originally placed. Frequently there is a scarcity of tissue of sufficiently good quality to close the defect satisfactorily. It is not surprising, then, that the hernia often recurs if the surgeon relies entirely on the stitching of a few muscle fibres, which in many instances are stretched and often extremely thin. In some of our cases careful suturing of the hiatus had been done elsewhere and they were operated on again in our clinic because of recurrence; at operation we found the hiatus wide and the crural fibres as if no suturing had ever been done.

With sliding hernias mere narrowing of the hiatus cannot therefore prevent recurrence. The other structural defects have to be corrected as well; the phreno-oesophageal ligament has to be repaired and the redundant hernial sac removed. In many techniques this is combined by an excision of the redundant sac and resuturing of the reduced portion to the diaphragm. As the phreno-oesophageal ligament arises from the underside of the diaphragm, this reconstruction is best performed by repairing the ligament on the abdominal side. Allison,[1] who uses the thoracic approach, makes a separate incision through the diaphragm to accomplish this. He incises the sac circumferentially and sutures the cuff, which remains attached to the oesophagus, to the underside of the diaphragm, to restore the phreno-oesophageal ligament. Madden[13] has stated that the cuff could be sutured to the top of the diaphragm with less technical difficulty and still accomplish the same result. This, however, does not anatomically restore the phreno-oesophageal ligament and cannot anchor the cardia *below* the diaphragm as well as in the technique employed by Allison. Neither can the alternative method of simply plicating the sac into the abdomen, as advocated by Sweet,[20] provide adequate anchorage.

Some authors have tried to fix the oesophagus itself to the narrowed hiatus with sutures passing through its wall. The oesophageal wall is weak, however, and not covered by peritoneum, so that it does not hold sutures well. Two of our cases where this repair had been done both had recurrence of the hernia soon after the operation.

It is clear, then, that only 2 of the defects can be repaired through the transthoracic route. The exposure of the hiatus is good and the redundant hernial sac can be adequately removed, but repair of the phreno-oesophageal ligament is impossible without entering the abdomen through a separate incision in the diaphragm. This renders the operation technically more difficult and increases the chances of post-operative complications.

The most important advantage of the abdominal approach is therefore the better approach to the phreno-oesophageal ligament. Another important advantage is that associated intra-abdominal disease can be dealt with. The simultaneous occurrence of hiatus hernia with gall-bladder diseases, for instance, is well known. In the series of 94 cases of hiatus hernia treated surgically, we performed cholecystectomy at the same time as the hernia repair on 23 occasions.

Perhaps, however, one of the greatest advantages of the abdominal approach is that it calls considerably less on the patient's reserves and leads to fewer post-operative pulmonary and cardiac complications. As many of these patients are elderly, the easier post-operative course is an important consideration.

All the structural defects can be best repaired through the abdominal route, and yet the recurrence rate of hiatus hernias repaired through this route remains high. This is probably because the material which has to prevent recurrence of the hernia is not of sufficient quality. Not only are the hiatal margins distorted and stretched and often thinned, but the phreno-oesophageal ligament, which is utilized as retentive structure, is often infiltrated with fat, stretched, and tenuous. If the sac is opened this structure becomes frayed and torn and has insufficient substance to hold sutures. We agree with Johnsrud,[12] who observes that 'the carefully prepared

cuff described by Allison ends up as a few shreds of peritoneum and areolar tissue'. These hernias, after all, are probably attributable to atrophy and relaxation of the muscle fibres surrounding the oesophageal opening, together with slackening of the elastic fibres of the phreno-oesophageal ligament.

The repair, then, is often not sufficiently strong to counteract the powers forcing the stomach back into the chest, i.e. the negative intrathoracic pressure, the positive intra-abdominal pressure, and the pull on the cardia exerted by the longitudinal muscle fibres of the oesophagus. Despite repair, the weakened structures of the hiatus often cannot prevent the cardia from slipping back into the posterior mediastinum. Thus one must look elsewhere for a means of securing the cardia in the abdomen.

To accomplish this we attempted gastropexy of the greater curvature to the left dome of the diaphragm, but 4 of the 7 sliding hernias we operated on in this way recurred. The diaphragm, a mobile muscular structure, is not ideal as a fixing point. Neither is there a suitable structure in the posterior abdomen to which the stomach can safely be attached. The idea was therefore conceived of suturing the stomach under tension to the anterior abdominal wall. Nissen advocated this for para-oesophageal hernia in 1955. Until 1956 he used the anterior surface of the stomach for fixation, suturing the greater curvature to the abdominal wall or costal margin on the left. He was sceptical about using this procedure for sliding hernias.

GASTROPEXY OF THE LESSER CURVATURE: RATIONALE

Since 1954 Boerema[4-6] has emphasized that in all types of hiatus hernia, but especially in sliding hernia, the *lesser curvature* should be employed for the gastropexy, since this part of the stomach constitutes the direct prolongation of the abdominal oesophagus. Only then can the upward pull on the cardia be efficiently counteracted, and only then is a sharp angle produced between the terminal oesophagus and the stomach fundus. This procedure gives very good results. The same effect cannot be produced by pexia of the anterior greater curvature and this explains its disappointing results in sliding hiatus hernias.

After gastropexy of the lesser curvature, the oesophagus, pulled down into the abdomen, will bend fairly sharply at the hiatus towards the anterior abdominal wall. When, in 1955, the technique of the operation was first described by Boerema and Germs,[5] it was called 'gastropexia geniculata anterior', to indicate the forward bend of the gastropexy (Figs. 10 and 11).

The high tension on the lesser curvature and the abdominal portion of the oesophagus prevents the cardia from sliding upwards into the mediastinum again. When the negative pressure in the thorax is increased during inspiration, the upward pull on the cardia is also increased so that the tendency to herniation becomes maximal. Simultaneously, however, the costal arch and upper anterior abdominal wall, to which the lesser curvature has been attached, are elevated so that the downward traction on the cardia becomes stronger (Fig. 13). Thus the greater tendency to herniation during inspiration is efficiently counteracted.

While reduction is maintained by the gastropexy, there is adherence between the narrowed hiatal wall and the denuded raw oesophageal surface. Thus the fibrous connection between the undersurface of the diaphragm and the oesophageal wall is restored, closing off the abnormal opening between abdomen and thorax.

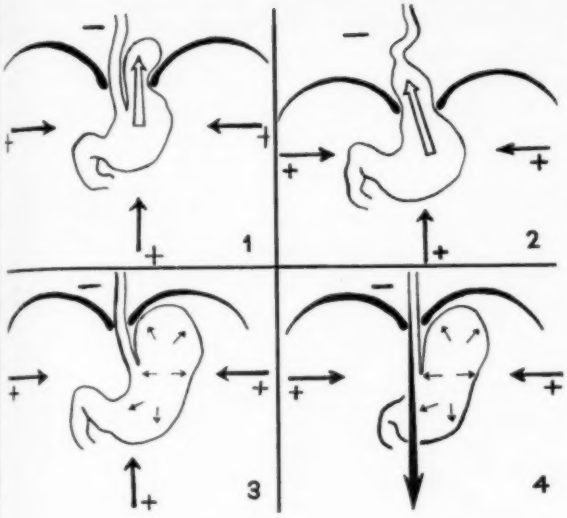
We are most satisfied with the anatomical results of the gastropexy. But even more important is the fact that all symptoms are relieved by the operation. This can be explained as follows:

With para-oesophageal hernia the complaints are produced by the pressure of an abnormal hernial mass in the thorax. The cardiac mechanism remains undisturbed. Reduction of the hernia and narrowing of the hiatus will prevent recurrence and relieve the symptoms completely. With sliding hernias, however, it is evident from the literature that no definite parallel exists between the success of anatomical repair and the relief of symptoms (Gertz *et al.*,[11] see above).

In our series, sliding hernias constitute by far the largest number of cases (87·8%). The majority of symptoms with these hernias are produced by incompetence of the cardiac mechanism and reflux of gastric contents into the oesophagus. This often leads to oesophagitis. Thus 29 of the 79 sliding hernias (more than one third) were associated with serious oesophagitis. It follows therefore that the success of an operation for sliding hernia will depend not only on an adequate anatomical repair, but also on correction of the disturbed cardiac mechanism. Recent investigations[2,3,7-9,21] indicate that the cardia can maintain its function independently of the diaphragm and that gastro-oesophageal closure depends on factors intrinsic to the cardiac region. Furthermore, as there is no oesophagitis or demonstrable reflux into the abdominal part of the oesophagus, the intrinsic mechanism must be situated at the gastro-oesophageal junction itself. These authorities agree that the final watertight closure between oesophagus and stomach is produced by mucosal folds at the cardia. These folds are approximated by an intrinsic muscular mechanism. Manometric and cineradiographic investigations have pointed to the presence of a segment of increased pressure at the cardia. These findings suggest that a sphincter exists which draws together the mucosal folds of the cardia, closing off the top of the stomach. It has not been conclusively demonstrated whether this sphincter depends for its action on circular muscle fibres in the terminal oesophagus or on the oblique muscle fibres of the stomach.

It seems clear, however, that gastro-oesophageal closure depends on both a valvular and a muscular mechanism. This mechanism normally prevents reflux of gastric contents into the oesophagus in all positions.

In para-oesophageal herniation the crural margins, which normally fit snugly around the oesophagus, become lax and widened and allow the herniation of a part of the stomach into the chest alongside the oesophagus. The hernia is forced into the chest through the widened hiatus whenever the patient bends forwards or increases the intra-abdominal pressure. This process is comparable to the way a 'blow-out' of the inner tube takes place through a defect in the outer tubing of a motor-car tyre. These hernias are not usually associated with reflux of gastric contents into the oesophagus. This is because the cardia remains in position below the hiatus and is never subjected to any gross increase of pressure on bending forwards or with exertion (Fig. 1). The closing mechanism can therefore maintain its function unhindered.



Figs. 1 - 4. See text, The hollow arrows in Figs. 1 and 2 indicate the flow of gastric contents.

Operative reduction of the hernia and narrowing of the widened hiatus restores normality.

With sliding hiatus hernias the cardia itself slides into the chest through the widened hiatus whenever the intra-abdominal pressure is increased or when the patient bends forwards. The gastric contents are then directed, as if into a funnel, against the herniated cardia (Fig. 2). The internal sphincter, whether it depends on the circular muscle fibres of the terminal oesophagus or on the oblique fibres of the stomach, is no longer able to approximate the mucosal folds and to close off the top of the stomach. The cardia becomes incompetent and reflux results. Thus reflux oesophagitis is the commonest complication of this type of hernia.

With the normal anatomy undisturbed, the pressure is equally distributed to all parts of the stomach so that the cardiac mechanism is not interfered with (Fig. 3). The most important aim, therefore, in the operative repair of sliding hiatus hernia is to create an abdominal oesophagus of sufficient length, thus restoring the normal pressure distribution in the stomach. The cardia, no longer situated at the apex of the hernia will then be able to resume its normal function.

This correction can only be achieved by the operation of gastropexia geniculata anterior (Fig. 4), where the oesophagus, after being mobilized in the hiatus, is pulled into the abdomen a distance of many centimetres, the lesser curvature then being attached under high tension to the anterior abdominal wall.

OPERATIVE TECHNIQUE OF GASTROPEXIA GENICULATA ANTERIOR (BOEREMA AND GERMS[4-8,10])

A median upper abdominal incision is made from the xiphoid process to the umbilicus, opening the abdomen through the linea alba. The xiphoid process is completely resected (Fig. 5). This improves the exposure and facilitates the approach to the hiatus, especially as many of the patients are rather obese. After preliminary palpation of the hiatus to confirm the diagnosis, the rest of the abdomen is thoroughly examined for other abnormalities, which are dealt with immediately or at subsequent operation, according to the nature of the pathology.

The triangular ligament of the liver is divided, so that the left lobe can be retracted out of the way towards the right, thus leaving a free approach to the hiatus (Fig. 5). The hernia is reduced by traction on the stomach and the hernial coverings incised transversely at the hiatus (Fig. 6). We prefer to use scissors for this procedure. The distal oesophagus with the two vagus nerves is then mobilized by blunt dissection. Traction on a thin rubber tube passed around the oesophagus facilitates the process of mobilization. The distal oesophagus is freed of its peritoneal and ligamentous attachments. The two vagus nerves are left intact. With this technique it is always possible to fully mobilize the gullet in the hiatus and to pull a few centimetres of thoracic oesophagus into the abdomen.

The crural fibres behind the oesophagus are now exposed. This process is facilitated by ligation and division of the upper branches of the left gastric artery, at the same time dividing the proximal attachment of the smaller omentum from the lesser curvature (Fig. 7). The cardia, thus mobilized, is pulled forwards and to one side. The crural fibres are carefully cleaned and then approximated behind the oesophagus with interrupted silk sutures (taking a rather bigger bite than is depicted in Fig. 8). As these sutures enclose muscle fibres, they should be tied lightly, working anteriorly towards the oesophagus. When the last suture is placed, the hiatus should still admit one finger alongside the gullet.

This concludes the actual repair of the hiatus. Superfluous hernial sac is removed but no further attempt is made to repair the sac or the phreno-oesophageal ligament. If the gall-bladder contains stones, or is obviously diseased, it is removed at this stage.

Before proceeding to the gastropexy, the surgeon should make sure that the nasal tube has entered the stomach. After gastropexy introduction of the tube is difficult owing to the sharp forward bend which the oesophagus makes at the hiatus.

The left liver lobe is returned to its normal position and the oesophagus and lesser curvature brought along its undersurface towards the anterior abdominal wall. This is done under strong traction so that some centimetres of thoracic oesophagus are pulled into the abdomen. The lesser curvature is then attached to the posterior rectus sheath at a point chosen to the right of the mid-line incision, fairly close to the right costal margin. While an assistant maintains strong traction on the stomach, the lesser curvature is stitched to the peritoneum and the posterior rectus sheath with 4 or 5 interrupted silk sutures. The abdominal portion of the oesophagus and its prolongation, the lesser curvature, should now form a tight cord, running straight forwards and somewhat caudal from the hiatus to the upper anterior abdominal wall (Fig. 9). This is the essential part of the operation and should be punctiliously performed.

After fixation of the lesser curvature, the stomach will fall backwards, the fundus coming to lie against the dome of the left diaphragm. In the past it was stitched into position here. Lately we consider this unnecessary, for after fixation of the lesser curvature the fundus spontaneously takes up its natural position next to the oesophagus, thus assuring the sharp angle of entry between oesophagus and stomach.

The abdomen is closed in the usual manner without drainage.

During the 12 years 1947 - 58, 94 patients with oesophageal hiatus hernia were operated on in Prof. I. Boerema's surgical clinic at the Wilhelmina Gasthuis, Amsterdam. The present study concerns the pre-operative clinical picture and the results of the various operative procedures employed.

CLINICAL FEATURES

Age. Only 7 of the 94 patients operated on were less than 30 years old; 4 of these were children under 10 years, the youngest being a baby of 9 months. In these children the hiatus hernia was probably due to congenital weakness of the hiatal structures. The majority of hernias (86%) occurred after the 4th decade, however, probably as a result of structural atrophy in the hiatal region; 13 patients were over the age of 70, the oldest being 79.

Sex. There was a striking preponderance in the female, the ratio of females to males being 3 : 1.

Symptoms

Symptoms are produced either by the presence of the abnormal space-occupying mass in the thorax, or by oesophagitis resulting from incompetence of the cardiac mechanism. Gastro-intestinal haemorrhage may cause a third symptom complex. Patients may also present with symptoms referred to distant organs along a variety of nerve pathways. In spite of the diversity of complaints, however, there is usually a definite pattern to the symptoms of hiatus hernia. Thus it is often possible on the history alone to base a strong suspicion of its presence.

Pain was the most constant single symptom, situated either in the epigastrium or under the lower sternum, or in both sites at once. In 72% of cases the patient complained of this type of pain, which was usually of a burning character and came on after meals. In 65% of cases there was a definite relationship to postural changes. Thus the pain was aggravated by lying down or bending forwards. Often patients woke up at night with pain, which was relieved by sitting up or walking about.

The presence of actual pain was denied by 13 of our patients, who preferred to speak of a discomfort in the epigastrium or substernally. This was experienced as a fullness after meals and was also affected by postural changes.

Lying down or bending forwards aggravated other symptoms as well. In 51% of cases, the patient complained of heartburn, which varied in intensity from mild to severe, when it amounted to substernal pain. Belching (35%) was a symptom experienced by many patients. It often relieved pain.

In 17% of our cases the patient complained of effortless regurgitation of fluid into the mouth on lying down or bending forwards. The fluid was sometimes tasteless, but more often sour-tasting and associated with heartburn. In 35% the patient complained of vomiting, usually of food just taken. As this was associated with nausea in only 14% of cases, it was often difficult to distinguish from effortless regurgitation, which indicates gross cardiac incompetence.

Another common symptom was dysphagia. One-third of our patients complained of a sensation as if food 'sticks' momentarily under the lower sternum. This symptom may be due to pressure of the herniated stomach on the oesophagus, or to oesophagitis, in the presence of which swallowing was often painful. Dysphagia was only severe or progressive in cases with associated ulceration or stricture.

Haemorrhage both manifest and occult, occurred from lesions in the herniated stomach or from oesophagitis, with or without ulceration. Haematemesis was present in 24% of patients, usually taking the form of streaks of blood in the vomitus. Severe haematemesis occurred occasionally. A history of recurrent melaena was given by 13% of our patients and 11% complained of dizziness, fainting spells and other typical symptoms of marked anaemia.

Four patients suffered from fits of coughing. In 2 of them, both in the 6th decade of life, the coughing occurred when lying down after meals; both had fits of coughing at night and in one case this was associated with burning substernal pain; in both cases it disappeared after repair of the hernia. The symptom can be explained by pressure of the hernial mass on the lungs or by a vagovagal reflex involving the pulmonary plexus. The other 2 cases with cough were infants, both of whom had chronic coughing which was probably due to the overflow of oesophageal contents into the bronchi.

Three of our cases had ear symptoms. An unpleasant itchy or painful sensation was experienced either in the left or right ear, usually coming on simultaneously with the abdominal symptoms in all cases. Earache[14] is explained as a referred pain along the auricular branch of the vagus nerve.

Three of our patients had been treated for angina pectoris elsewhere before being admitted for repair of the hernia. Only one of these was relieved by the operation; her pre-operative electrocardiogram had shown no signs of ischaemia, and the angina-like symptoms were attributed to a large, irreducible, sliding hernia. Both the other patients, although relieved of all abdominal symptoms, still require treatment for angina pectoris, and in these two cases one must accept the diagnosis of both coronary artery disease and hiatus hernia; one of them had mild signs of ischaemia on the ECG.

Duration of symptoms. Of the 94 patients, more than half (56%) had symptoms of at least 5 years' duration. In this group, 18 had complaints of more than 20 years standing, of whom 3 said they had suffered from symptoms for more than 35 years.

Clinical Examination

General examination, apart from signs of anaemia in some cases, presented no diagnostic features. There were also few abdominal signs. Occasionally the upper abdomen was slightly tender on palpation, but otherwise the clinical examination was usually negative.

Laboratory Examination

The haemoglobin, white blood cells and erythrocyte sedimentation rate were determined pre-operatively in all cases, as well as a fractional test meal and occult blood test. Haemorrhage was frequent, both manifest and occult. Of our patients 11% complained of symptoms due to marked anaemia and 15% had a haemoglobin level of less than 11 g.%. Many of these patients required repeated blood transfusion to bring the haemoglobin back to normal level. Four had a haemoglobin level of less than 7·8 g.%.

A mild leucocytosis, not exceeding 12,000 per c.mm., was present in only 5 of our patients. In 1 case this may have been caused by associated gall-bladder disease. None of these 5 patients suffered from oesophagitis. It is important to note that, contrary to what some authorities have stated, we found no leucocytosis in any of our 29 cases of serious oesophagitis.

There were only 8 patients with a slightly raised ESR, of whom 3 had concomitant gall-bladder disease. Only 2 of the 29 cases with marked oesophagitis had a raised ESR.

Of the 94 patients, there were 26 with occult blood in the faeces. Of these, 50% had demonstrable oesophagitis. The remainder probably had bleeding from a lesion in the herniated stomach.

Fractional test meal showed the total acid to be raised above 70 (c.c. N/10 HCl) and the free acid above 40 (c.c. N/10 HCl) in 22·4% of cases. Of the patients with oesophagitis, only 25% had a raised level of acidity. Hyperacidity is not therefore an essential factor in reflux oesophagitis. One patient, a woman of 74 years, with a sliding hernia associated with free reflux, had severe oesophagitis and yet her total acid amounted to only 10, and she had no free HCl.

Radiological Examination

The diagnosis was confirmed by radiological examination in the Trendelenburg position. There were no cases of true congenitally-short oesophagus (in all cases the cardia could be reduced below the hiatus at operation). By far the largest number of patients, viz. 87·8%, had sliding hernias, 6·7% para-oesophageal hernias, and 5·5% hernias of the mixed variety.

Oesophagoscopy

Oesophagoscopy was not performed in every case. Its value as a means of detecting oesophagitis was at first not fully realized. Where the X-ray diagnosis was clear, no oesophagoscopy was done, even in the case of associated haemorrhage or other complications. Lately it has become evident that oesophagitis should be treated pre-operatively in order to prevent complications during operation and to increase the possibility of a successful repair. In most of the later cases, therefore, oesophagoscopy was performed, particularly in those cases where the history suggested oesophagitis or where occult blood was present in the faeces.

In the series of 94 cases oesophagoscopy was performed 45 times and marked oesophagitis diagnosed in 29 cases. All these patients showed severe erythema, and bleeding on mild trauma, and oedema of the mucous membrane. Cases with mild oesophagitis, where there is only mild erythema and where the changes seen are often very slight and only recognized by the very experienced endoscopist, are not included. Of the 29 patients with severe oesophagitis, many showed diffuse superficial ulceration; 2 of them had chronic oesophageal ulceration, in both of which a chronic ulcer niche was seen at the cardia, the diagnosis of benign chronic inflammation confirmed by biopsy; and the ulcer was palpable at operation.

The height of the cardia varied from 30 cm. to 40 cm. One patient, a woman aged 55 years with a large hiatus hernia, had a cardia situated at 27 cm. Occasionally the cardia was encountered at a normal level in patients in whom definite herniation was shown

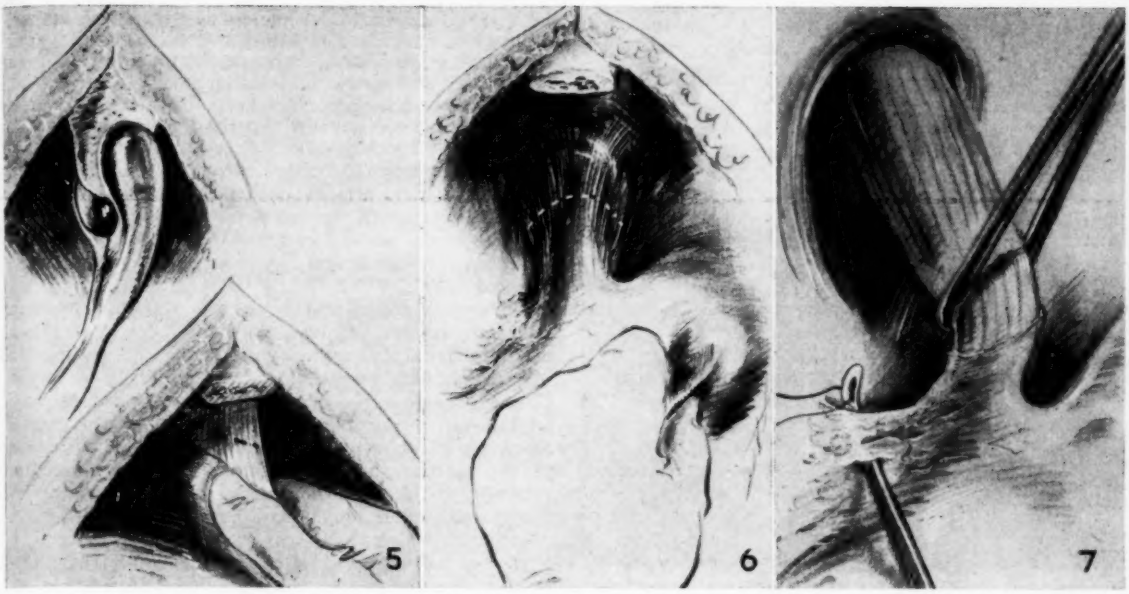


Fig. 5. Median upper abdominal incision. Xiphoid process resected and triangular ligament of left liver lobe divided.
Fig. 6. The left liver lobe has been retracted to the right. Stomach retracted downwards. Stretched peritoneum and phreno-oesophageal ligament incised transversely at the hiatus.
Fig. 7. Oesophagus mobilized in the hiatus. Smaller omentum ligated and divided until upper lesser curvature is quite mobile.

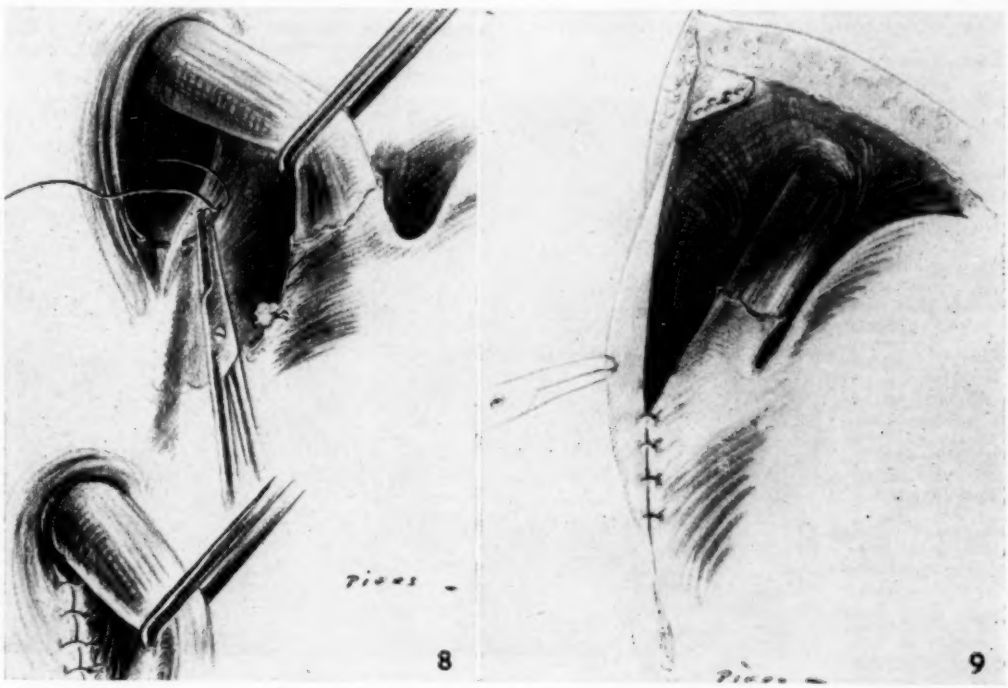
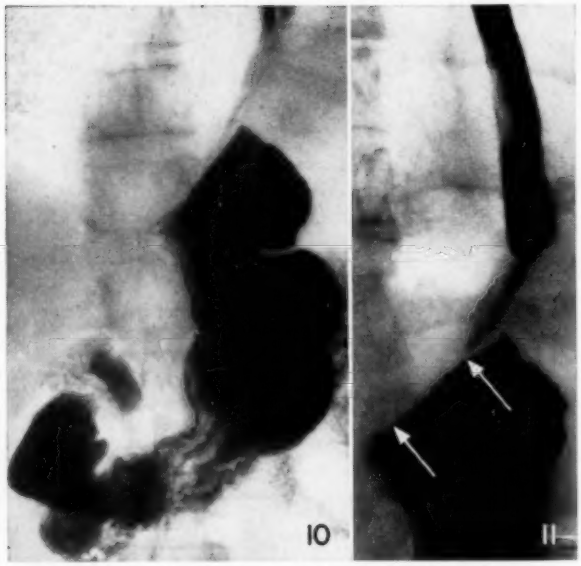


Fig. 8. Fibres of right crus approximated behind the oesophagus with interrupted silk sutures.
Fig. 9. Lesser curvature attached under high tension to the anterior abdominal wall, the sutures passing through peritoneum and posterior rectus sheath.

Figs. 5 - 9 drawn by Piers.



Figs. 10 *and* 11. Antero-posterior views after gastropexia geniculata anterior. Arrows indicate line of attachment of lesser curvature to anterior abdominal wall. Note length of intra-abdominal oesophagus and forward bend at the hiatus.

by X-ray. This may be because the stomach was not herniated at the time of examination or because the cardia was pushed down in front of the oesophagoscope. Nevertheless, a cardia situated at an abnormally high level confirms the diagnosis, especially when associated with free reflux of gastric juice into the oesophagus.

Pre-operative Treatment

All cases operated on were given pre-operative breathing exercises and, whenever possible, the patient was kept ambulant till the day of operation. Where complications were present, special pre-operative treatment was given.

In cases of sliding hernia associated with severe oesophagitis and ulceration, the patients were treated pre-operatively with a continuous milk-alkali drip through a Ryle's tube left in the proximal oesophagus. This bathes the inflamed oesophagus with alkaline fluid, while neutralizing the gastric contents that are regurgitated.

Patients with gross anaemia were given pre-operative blood transfusion.

OPERATIVE RESULTS

All the patients in this series were called up for clinical and radiological re-examination not less than 6 months after the operations; in the majority (86%) the interval was more than a year. Most authorities have pointed out that recurrences, when they do occur, are manifest immediately after operation, and this has been borne out by our series.

Various operative procedures were followed in this series and the hernia repaired by both the transthoracic and the transabdominal routes. In 3 patients operated on transthoracically symptoms recurred immediately after operation. Poor results were also obtained in 17 cases in which the hiatus was narrowed through the abdominal approach and the oesophagus secured to the hiatal rim or the stomach attached to the diaphragm. Of these 17 patients, symptoms recurred in 59%.

The results of gastropexia geniculata anterior were far more satisfactory. Only 4 (5·2%) of the cases had clinical and radiological recurrence of the hernia. This was manifest immediately after operation, the symptoms often returning before discharge and always within the first few post-operative weeks when the patient resumes normal duties. Another 5 patients, on follow-up examination, said they were improved by the operation but not symptom-free; none showed recurrence of the hernia on repeated examination in the Trendelenburg position, but 2 of the 5 cases still had gastro-oesophageal reflux.

We found 67 of the 77 patients operated on by the method of gastropexia geniculata anterior completely symptom-free on follow-up examination. All these patients showed neither recurrence nor reflux on radiological re-examination. The operation was therefore a complete success in 87% of cases treated by this method. On radiological examination the cardia was always seen to lie well below the hiatus (Figs. 10-13). On the lateral X-rays the abdominal oesophagus and lesser curvature appeared tautly stretched to the anterior abdominal wall. There was a marked difference between expiration and inspiration. During inspiration when the

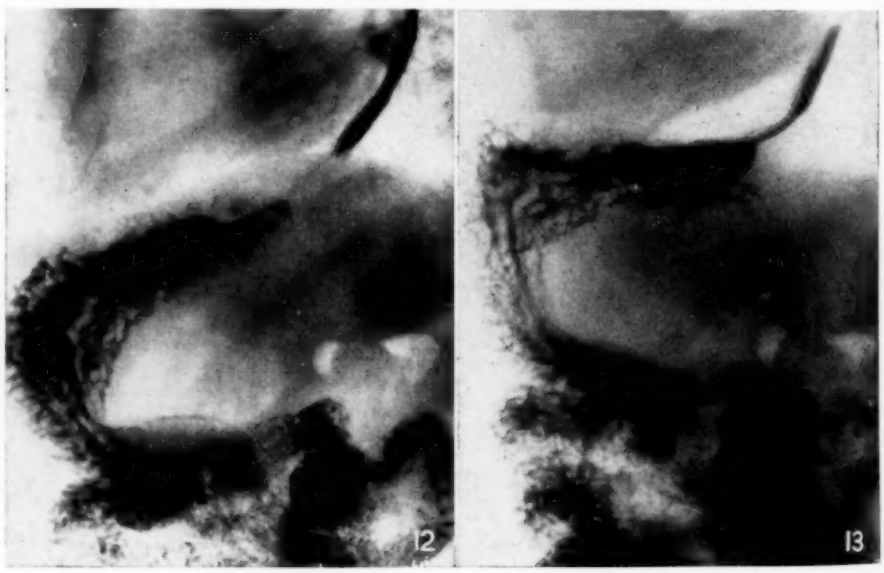


Fig. 12. Lateral view of patient after gastropexia geniculata anterior during expiration.

Fig. 13. The same patient during inspiration. Note that the lesser curvature is drawn upwards and forwards. This efficiently counteracts the greater tendency to herniation during inspiration.

tendency to herniation is usually greatest, the lesser curvature was pulled even more taut by the elevation of the costal margin and upper abdomen (Fig. 13). The cardia remained competent in the Trendelenburg position, also on increase of intra-abdominal pressure.

Both the anatomical deformity at the hiatus and the disturbed physiology of the cardia are therefore restored by the operation.

As the procedure is a transabdominal one, it is possible to deal with concomitant abdominal pathology. Cholecystectomy was performed simultaneously with the hernial repair by gastropexia in 32% of cases.

There was one post-operative death, giving a mortality of 1·3%. The post-operative course was otherwise smooth and no complications of a serious nature occurred.

The results of gastropexia geniculata anterior are therefore most satisfactory when compared to those of other techniques employed in this series and the results quoted in the literature.

SUMMARY

The anatomical and physiological disturbances associated with oesophageal hiatus hernia are discussed as they apply to surgical treatment. It is concluded that the abnormal hiatal anatomy and disturbed function of the cardia can only be adequately restored by gastropexy of the lesser curvature to the anterior abdominal wall according to the technique of gastropexia geniculata anterior after Boerema and Germs.

This is borne out by a study of 94 cases of oesophageal hiatus hernia treated surgically. Whereas only 35% of the 17 cases treated by other operative methods were symptom-free on follow-up examination, the results obtained by gastropexia geniculata anterior were most satisfactory. Clinical and radiological follow-up examination showed the operation to be a complete success in 87% of the 77 patients treated by this method.

I wish to thank Prof. I. Boerema, head of the Department of Surgery at the Wilhelmina Gasthuis, Amsterdam, for his encouragement and advice.

REFERENCES

1. Allison, P. R. (1951): Surg. Gynec. Obstet., **92**, 419.
2. Atkinson, M., Edwards, D. A. W. and Rowlands, E. N. (1957): Lancet, **2**, 918.
3. Atkinson, M., Edwards, D. A. W., Honour, A. J. and Rowlands, E. N. (1957): *Ibid.*, **2**, 1138.
4. Boerema, I. and Germs, R. (1955): Arch. chir. neerl., **7**, 351.
5. *Idem* (1955): Zbl. Chir., **80**, 1585.
6. Boerema, I. (1958): J. Int. Coll. Surg., **29**, 533.
7. Botha, G. S. M. (1958): Brit. J. Surg., **45**, 569.
8. Braasch, J. W. and Ellis, E. H. (1956): Surgery, **39**, 901.
9. Dornhorst, A. C., Harrison, K. and Pierce, J. W. (1954): Lancet, **1**, 695.
10. Germs, R. (1954): Ned. T. Geneesk., **98**, 1630.
11. Gertz, T. C., Regout, J. E. P. M. and Thomsen, G. (1951): Thorax, **6**, 316.
12. Johnsrud, R. L. (1956): Surg. Gynec. Obstet., **103**, 708.
13. Madden, J. L. (1956): *Ibid.*, **102**, 187.
14. Malherbe, W. D. F. (1958): Lancet, **1**, 1368.
15. *Idem* (1959): Acad. thesis, University of Amsterdam.
16. Nissen, R. (1955): Dtsch. med. Wschr., **80**, 467.
17. Nissen, R. (1956): *Ibid.*, **81**, 185.
18. Nissen, R. (1956): Amer. J. Surg., **92**, 389.
19. Nissen, R. (1957): *Lehrbuch der Chirurgie*, Zwerchfell, p. 529. Stuttgart: Georg Thieme Verlag.
20. Sweet, R. H. (1952): Ann. Surg., **135**, 1.
21. Ton, J. G. (1958): *Reflux Oesophagitis*. Amsterdam: Born.

SOUTHERN AFRICAN CARDIAC SOCIETY (CAPE WESTERN REGION)

A meeting of the Southern African Cardiac Society (Cape Western Region) was held in the Department of Medicine, University of Cape Town, on 9 June 1960 at 8.15 p.m. Five papers were presented on current research in ischaemic heart disease by a panel of speakers consisting of Drs. B. Bronte-Stewart, M. C. Botha, M. Fewster, L. Krut, I. Bouchier, and Messrs. V. Wells and J. Wilkens.

1. BLOOD GROUP PATTERN IN ISCHAEMIC HEART DISEASE

It is not easy to differentiate those factors contributed by heredity from those occurring in the environment, and the extent to which the one is overborne by the other becomes a vital issue with respect to ischaemic heart disease. There is much in favour of the view that susceptibility to ischaemic heart disease may be inherited, at least in part; but as yet there are no studies that can be accepted as conclusive in this regard. The blood groups of 508 White patients with ischaemic heart disease have now been determined and a significantly different pattern is seen when this is compared with the blood donors' pattern in the Western Province. In the patients there was a preponderance of group A and a lower frequency of group O than in the controls, and this was seen at each decade of age and in both sexes. Even in the small Cape Coloured sample which was grouped, a preponderance of group A was seen in those suffering from ischaemic heart disease. On the results of this investigation it could be calculated that the probability of a group A individual having ischaemic heart disease was almost one-and-a-half times as great as a group O individual. There was therefore strong evidence of a hereditary factor in the development of ischaemic heart disease. It was, however, emphasized that certain epidemiological trends could only be explained on the basis of additional environmental influences, which was fortunate since it brought great hope that the increasing mortality and morbidity of the disease could be controlled.

2. PLASMA FATTY ACIDS AND ATHEROSCLEROSIS

A survey has been undertaken to study the plasma lipid composition of patients with coronary disease, of a European control group, and of Bantu males between the ages of 40 - 50 years. Separation of the cholesterol ester and triglyceride fractions of the total and beta-lipoprotein fractions of the plasma has been carried out by silicic-acid chromatography after heparin precipitation. Subsequent separation, detection, and evaluation of the percentages of the component fatty acids by the highly sensitive technique of gas-liquid chromatography, indicates that under the conditions of the investigation there is little difference between the groups. Despite marked differences in their mean plasma-cholesterol levels, there appears to be a characteristic fatty-acid pattern in the plasma fractions which the body tries to maintain and which is reflected in the fatty-acid pattern of corresponding fractions of 9 atheromatous plaques studied concurrently. The high proportion of linoleic acid in the cholesterol esters of the plasma and plaques did not support the concept that deposition is intensified or accelerated in the absence of essential fatty acids or in the presence of more saturated fatty acids in the cholesterol ester.

3. DEPOT FAT COMPOSITION IN THE THREE RACIAL GROUPS AND CORONARY SUBJECTS

Depot fat composition reflects the nature of the diet and particularly its fat content; this being more representative of intake over prolonged periods of time than is the serum. Depot fat, unlike serum, is almost entirely triglyceride. Adipose tissue has been shown to be a metabolically highly active 'organ' and can be sampled painlessly and easily by needle biopsy. An analysis of the composition of the depot fats in the 3 local racial groups, males and females, at two age levels and in coronary subjects was studied by gas-liquid chromatography. The same fatty acids were demonstrated in all subjects. It was demonstrated that while there were differences in the proportion of individual fatty acids between the groups, there were wide differences for each fatty acid within the groups. The differences between the groups are thus probably not significantly different.

4. FAT TOLERANCE STUDIES IN ISCHAEMIC HEART DISEASE

In ischaemic heart disease the patients exhibit a more extensive and more prolonged lipaemia than controls following a standard fat meal. The reason for this is obscure. Previous studies have revealed that differences in fat clearing do not exist between the

patients and controls. In this study no differences were found between the races in their tolerance to a standard fat meal provided that they did not have ischaemic heart disease. Fat, fed orally, showed significant differences between patients and controls but no differences were seen when the same fat was administered intravenously. The obvious conclusion is that the differences arise from absorptive mechanisms. As a possible lead for further study, the marked differences in fat tolerance between patients having treatment with and without antibiotics were shown.

5. ACTIVE PRINCIPLES IN EGG-YOLK LIPID

In spite of the relatively high iodine value (70) egg-yolk lipids produce a higher serum cholesterol, gram for gram, than any other known fat. To study this phenomenon egg yolks were fractionated into their various chemical components which were then fed to a human subject. The active principle was found in the acetone-soluble component. Following saponification and the feeding of the unsaponifiable and the saponifiable fractions separately, no rise in serum cholesterol was seen. If fed together, however, a rise equivalent to that seen on feeding the parent egg yolk, occurred. If fed 6 hours apart, only an insignificant rise was seen. The experiments prove conclusively that both cholesterol and the type of fatty acids play an important part in determining the effects produced. A theory has been proposed according to which cholesterol is absorbed by the body only if in solution in accompanying dietary fat. The serum-cholesterol regulating properties can thus be related to the solubility of dietary or endogenous cholesterol in these fats.

These studies were supported in part by research grants from the National Heart Institute, USA (PHS: H-3316) and the South African Council for Scientific and Industrial Research.

MEDICAL ASSOCIATION OF SOUTH AFRICA

ANNUAL REPORT OF THE CHAIRMAN OF FEDERAL COUNCIL FOR THE YEAR ENDED 30 JUNE 1960

Obituary. It is with deep regret that we record the loss through death of the following members: Drs. G. W. Brammer, C. T. I. Clarke, K. O'L. Doherty, F. H. Dommisse, G. W. Doran, L. du Preez, M. C. A. Erasmus, P. A. Euvrard, H. Evans, M. I. Futerman, E. L. Galgat, E. S. Gray, M. Hoffman, D. Hugo, M. H. Jacobs, J. Kingsley, G. N. S. Klooster, A. E. Laubscher, B. Leader, D. R. Lee, K. H. Lepehne, J. N. W. Loubser, A. P. Martin, F. H. Molliere, P. K. Morrison, J. T. McGinn, E. J. Papenfus, J. Pratt-Johnson, S. M. Ribane, H. Roseman, S. T. Seccombe, D. T. Shanks, E. Song, J. D. Strachan, A. A. van den Heever, D. Vollet, and M. J. M. Wolfaard.

Membership. During the past year there has been an overall increase in members of 15, the total membership now being 5,517. In addition there are 79 student members. Members are distributed as follows: Border Branch 213, Cape Eastern Branch 56, Cape Midland Branch 228, Cape Western Branch 1,269, Eastern Transvaal Branch 276, Goldfields Branch 75, Griqualand West Branch 99, Natal Coastal Branch 561, Natal Inland Branch 193, Northern Transvaal Branch 551, O.F.S. and Basutoland Branch 307, Southern Transvaal Branch 1,191, South West Africa Branch 74, Transkei Branch 76, Vaal River Branch 59, unattached members 251, emeritus members 30, and honorary members 8. There is no doubt that our membership should be greater than it is and that a large number of practitioners are remaining outside the Association while benefiting as a result of its work.

Honours. During the year under review the Council agreed to the award of the Association's Silver Medal for distinguished service to medical science and humanity to Dr. James Gear, present Director of the South African Institute for Medical Research, in recognition of his considerable contributions to research notably in connection with poliomyelitis. Bronze medals for meritorous service to the Association were awarded to Dr. C. Adler (Southern Transvaal Branch), Dr. H. Grant-Whyte (Natal Coastal Branch) and Dr. E. Meltzer (Eastern Transvaal Branch). In addition Council honoured Prof. R. Dart, Dr. G. Buchanan, Mr. I. W. Brebner, Dr. J. Pratt-Johnson and Dr. B. Weinbren, by electing them to emeritus membership.

Annual General Meeting. The Annual General Meeting of the Association for the year 1959 took place in East London on 24 September 1959. At the conclusion of the formal business, Dr. P. F. H. Wagner was inducted as President by Dr. R. Schaffer, the retiring President. The meeting was then adjourned until the following Monday evening when, combined with the opening ceremony of Congress, Dr. Wagner delivered his Presidential Address and awards were presented. This was followed by a reception.

Congress. The 42nd South African Medical Congress was held at East London from 27 September to 3 October 1959 and proved to be a very successful function. The Association was honoured by the presence of certain distinguished overseas guests and is grateful to the Border Branch which was responsible for the organization on this occasion.

Federal Council. There have been 2 meetings of the Council during the year under review. The first of these was held in East London on 24 - 26 September 1959 immediately preceding the Congress and the second in Pretoria on 3 - 5 March 1960. The average attendance was 53 out of a total membership of 60. The Executive Committee met on 4 occasions. Two meetings were held on the day preceding the Council meetings and 2 special meetings were held in Pretoria in July and December 1959.

Committees of Council

The Head Office and Journal Committee continues to play its part in the administrative and financial affairs of the Association and its Journals. At the Council meeting in March, Dr. R. Kleinman was appointed to be Assistant Editor as from 1 July 1960. Dr. T. Shadick Higgins, who had assisted as Associate Editor after the appointment of Dr. Blignault to the editorial chair, retired on 30 June after over 7 years of service to the Journals. The Association is grateful to Dr. Higgins for his untiring efforts and his outstanding contribution to its work.

The Federal Ethical Committee has not had occasion to meet nor have any matters been referred to it for attention.

The Central Committee for Contract Practice has continued to do a considerable amount of routine work and has dealt with a number of new applications for recognition as approved medical aid societies. Towards the end of 1959 Dr. Marchand, Associate Secretary of the Association, whose main responsibility has been contract practice affairs, was transferred to the Pretoria Office.

The Parliamentary Committee has had several meetings and has dealt with a large amount of business, most of which has been concluded with advantage to members of the Association.

The Workmen's Compensation Act Committee has continued to act in liaison with the Commissioner and has attended to a number of routine matters.

The Medical Insurance Committee was appointed in September 1959 to consider the problems arising from the entry of certain life insurance companies into the medical aid field. The committee met representatives of the two companies mainly concerned, members of the board of the Medical Services Plan which had recently been established and other interested persons. As a result it recommended, and Council agreed at the March 1960 meeting to recognize certain 'groups' within the medical insurance schemes which conform to the rules for approved medical aid societies laid down by the Association. Negotiations with the companies concerned are proceeding.

Journals. Control of the number of text pages in the Association's weekly *Journal* has led to some financial improvement but the demand for space continues and there is no lack of articles of a high standard which should be published. The quarterly *South African Journal of Laboratory and Clinical Medicine* continues to be published and fulfils a definite function in providing a vehicle for the more specialized article.

Branches, Divisions and Groups continue to hold regular meetings, to serve the members resident in their areas, and to promote their special interests. A new branch, the Orange

Free State Goldfields Branch, was recognized as from 1 January 1960 with headquarters at Welkom. Once again it should be stressed that, although business meetings may be important, the emphasis should be placed on clinical meetings.

World Medical Association. The Thirteenth General Assembly of the World Medical Association was held in Montreal, Canada, in September 1959 and on that occasion we were represented by ex-members who are now resident in Canada, Prof. Harding le Riche and Dr. D. A. van Binnendyk. The Second World Conference on Medical Education, convened by the World Medical Association, was held in Chicago prior to the General Assembly and our representative was Mr. T. B. McMurray. It is becoming increasingly important that South Africa should take its part in world affairs and that we should be represented on all occasions by a formal delegation rather than having to rely on persons who may be in the area at the time of the meeting. For this reason it is desirable that adequate provision be made in our budget for this purpose. South Africa cannot afford to lag behind the other African states in this regard.

Commonwealth Medical Conference. This was held in London in July 1959. The President, Dr. R. Schaffer, who was our official delegate at the Annual Meeting of the British Medical Association in Torquay, attended the Conference as our representative.

Finance. With the raising of the subscription it was estimated that the loss of the previous year of £8,805 would be converted into a small surplus. This surplus eventually proved to be £922. With the losses which the Association has sustained over the last few years, it will take many years at this rate to recover our previous capital and the costs of all commodities continue to rise.

Benevolent Fund. The fund has made provision for 32 beneficiaries during the year, 1 of whom recently died. The accumulated funds stood at £51,981 on 31 December 1959 and during the financial year grants totalling £4,592 17s. 3d. were made. The Council is grateful to the many who have contributed to the fund and especially the various Medical Wives Auxiliary Committees who have done so much in this regard.

Library grants. Grants totalling £800 have been made to the libraries of the 5 medical schools during the year under review.

Medical agencies. The agencies maintained in Cape Town and Johannesburg continue to render valuable services to members, who are reminded that the agencies were established to be of service to all who require assistance. The *Medical Insurance Agency* also provides a service to members through various forms of insurance — public liability, motor car, life and endowment and the many forms of general insurance. A recent addition has been the establishment of the Medical Association of South Africa Retirement Annuity Fund under the provisions of the Income Tax Act (No. 58 of 1960). A number of members have joined so that their membership will date from 1 June 1960. When final details have been worked out, all members will be informed through the medium of the *Journal* and by circular.

Conclusion

On behalf of the Council I would thank all who have contributed to the work of the Association. I refer particularly to the honorary officials and members of the various Committees of Council and of the Branches, Divisions and Groups. Many of these officials have made great sacrifices of time and leisure in the interests of the Association and of their colleagues in the profession.

I would also extend the thanks of the Council to members of the staff, both medical and lay, who have served the Association well.

J. H. Struthers
Chairman of Council

Pretoria
15 July 1960

SUID-AFRIKAANSE GENEESKUNDIGE EN TANDHEELKUNDIGE RAAD : SOUTH AFRICAN MEDICAL AND DENTAL COUNCIL

WYSIGING VAN DIE REËLS BETREFFENDE GEDRAG WAARVAN DIE RAAD KENNIS KAN NEEM

Goewermentskennisgewing No. 991, van die Departement van Gesondheid, word hieronder herhaal vir die inligting van lede van die Vereniging:

„Die Minister van Gesondheid het, in die uitoefening van die bevoegdheid hom verleen by subartikel (4) van artikel *vier-en-negentig* van die Wet op Geneeshere, Tandartse en Aptekers, 1928 (Wet No. 13 van 1928), sy goedkeuring geheg aan die wysiging van die reëls betreffende gedrag waarvan die Raad kennis kan neem, soos opgestel deur die Suid-Afrikaanse Geneeskundige en Tandheelkundige Raad kragtens subartikel (2) van genoemde artikel van die Wet en afgekondig by Goewermentskennisgewing No. 49 van 11 Januarie 1946, soos gewysig:

„*(a)* Deur die bestaande reël 13 te skrap en deur die volgende te vervang:

„13. *Konsultante en Spesialiste.*

(1) Die handelings verbied in (of die late ten opsigte van handelings vereis ingevolge die bepalings van) die reëls afgekondig kragtens paragraaf (*r*) van subartikel (2) van artikel *vier-en-negentig* van die Wet op Geneeshere, Tandartse en Aptekers, 1928 (Wet No. 13 van 1928), soos gewysig, te wete die reëls waarin die voorwaardes beliggaam is betreffende die praktyk van geneeshere en tandartse wie se spesialiteite geregistreer is.

(2) Hom as 'n konsultant voordoen wanneer hy pasiënte behandel, behalwe in oorlegpleging met en op versoek van ander praktisyns."

„*(b)* Deur die volgende nuwe reël in te voeg:

„14 *bis. Verhindering van 'n pasiënt op 'n onbetaamlike wyse.*

Verhindering op 'n onbetaamlike wyse van 'n pasiënt (of 'n persoon wat behoorlik namens die pasiënt optree) wat die mening van of behandeling deur 'n ander praktisyn verlang".'

AMENDMENT OF THE RULES REGARDING CONDUCT OF WHICH THE COUNCIL MAY TAKE COGNISANCE

Government Notice No. 991, from the Department of Health, is reproduced below for the information of members of the Association:

'The Minister of Health, in exercise of the powers conferred on him by sub-section (4) of section *ninety-four* of the Medical, Dental and Pharmacy Act, 1928 (Act No. 13 of 1928), has approved of the amendment of the rules regarding conduct of which the Council may take cognisance, made by the South African Medical and Dental Council under sub-section (2) of the said section of the Act, and published under Government Notice No. 49 of 11th January, 1946, as amended:

'*(a)* By the deletion of the present rule 13 and the substitution therefor of the following:

"13. *Consultants and Specialists.*

(1) The commission of acts prohibited in (or the omission of acts required in terms of) the rules promulgated under paragraph (*r*) of sub-section (2) of section *ninety-four* of the Medical, Dental and Pharmacy Act, 1928 (Act. No. 13 of 1928), as amended, namely the rules embodying the conditions governing the practice of medical practitioners and dentists whose specialities have been registered.

(2) Holding himself out as a consultant when attending and treating patients except in consultation with and at the request of other practitioners."

'*(b)* By the insertion of the following new rule:

"14 *bis. Improperly impeding a patient.*

Improperly impeding a patient (or a person acting on behalf of a patient) who desires to obtain the opinion of or treatment by another practitioner".'

IN DIE VERBYGAAN : PASSING EVENTS

South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 15 August at 5.10 p.m. in the Institute Lecture Theatre. Dr. H. J. Heinz will speak on 'Epidemiology of ancylostomiasis in the Kalahari Bushman'.

* * *

University of Cape Town and Association of Surgeons of South Africa (M.A.S.A.), Joint Lectures. The next lecture in this series will be held on Wednesday, 10 August, at 5.30 p.m. in the E-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. Mr. P. J. M. Retief will speak on 'Urology overseas'. All members of the Medical Association are welcome to attend this lecture.

* * *

Dr. J. H. S. Gear, Director of the South African Institute for Medical Research, Johannesburg, has been invited to attend the Fourth Conference of the Industrial Council for Tropical Health at Boston and the Fifth International Poliomyelitis Conference in Copenhagen, and left for overseas on 17 July. At the tropical health conference, Dr. Gear will be Chairman of the section on schistosomiasis and will read a paper on the problem of bilharziasis in South Africa.

At the Poliomyelitis Conference, Dr. Gear will officially represent the South African Institute for Medical Research and the University of the Witwatersrand, and will be a panel member in one of the sections.

* * *

Dr. R. E. Bernstein, Head of the Electrolyte Research Unit, South African Institute for Medical Research, Johannesburg, has left for overseas, where he will attend 5 international congresses at which subjects related to his research activities will be discussed. They are the First International Congress of Endocrinology, Copenhagen, 18 - 23 July; the Fourth International Congress of Clinical Chemistry, Edinburgh, 14 - 19 August; the Sixth International Congress of Internal Medicine, Basel, 24 - 27 August; the First International Congress of Nephrology, Geneva and Evian, 1 - 3 September; and the Tenth Congress of the International Society for Cell Biology, Paris, 4 - 9 September.

Dr. Bernstein will be Chairman of the section on 'Metabolic abnormalities' and leader of a discussion group at the International Congress of Clinical Chemistry in Edinburgh. He will read papers at 4 of the abovementioned congresses. These papers will deal with various aspects of Dr. Bernstein's research into the metabolism of red cells, estimation of body water with amino-antipyrines, water diuresis, and cation transport by blood cells.

Dr. Bernstein has been awarded an S. L. Sive Memorial Travelling Fellowship to enable him to attend these congresses.

Mr. Ben Bellon, ear, nose and throat surgeon, has recently returned to Johannesburg after a 3 months' visit to otological clinics in Europe and England.

Dr. Ben Bellon, oor-, neus- en keelarts, het onlangs na Johannesburg teruggekeer na 'n besoek aan otologiese klinieke op die Vasteland en Engeland.

* * *

Dr. B. M. Kennelly, of Cape Town, has been awarded a Commonwealth Scholarship for postgraduate study and research in medicine at the University of Edinburgh.

* * *

South African Paediatric Association (M.A.S.A.), Natal Sub-Group. A meeting of this Group will be held on Wednesday 17 August at 8 p.m. at the Durban Medical School. Dr. M. K. Hathorn will speak on 'Iron metabolism — with particular reference to infants and children'.

* * *

Drakenstein Division (M.A.S.A.). The next meeting of this Division will be held on Friday 12 August at 7.30 p.m. at the Central Hotel, Paarl, Cape. Mr. J. D. Joubert, urologist, will speak on 'Urology in childhood'.

Afdeling-Drakenstein (M.V.S.A.). Die volgende vergadering van hierdie Afdeling sal op Vrydag 12 Augustus in die Central Hotel, Paarl, Kp., om 7.30 nm. gehou word. Dr. J. D. Joubert, uroloog, sal oor ‚Urologiese siektes by kinders' praat.

* * *

Lede word daaraan herinner dat hulle die Sekretaris van die Mediese Vereniging van Suid-Afrika, Posbus 643, Kaapstad, sowel as die Registrateur van die Suid-Afrikaanse Geneeskundige en Tandheelkundige Raad, Posbus 205, Pretoria, moet verwittig van enige adresverandering. Versuim hiervan beteken dat die *Tydskrif* nie afgelewer kan word nie. Dit het betrekking op lede wat oorsee gaan sowel as dié wat binne die Unie van adres verander.

* * *

Prof. A. J. Brink, van die Universiteit van Stellenbosch en die Karl Bremer-hospitaal, wat saam met dr. J. H. Cairns, van die Kaapse Provinsiale Administrasie, op 'n uitgebreide oorsese studietoer was, het onlangs teruggekeer en sy werksaamhede hervat.

Professor Brink en dr. Cairns het hospitale in die V.S.A., Kanada, die V.K., en op die Vasteland van Europa besoek. Die doel van hul sending was om 'n spesiale studie te maak van hospitaalbeplanning, met verwysing na die voorgenome bou van die nuwe opleidingshospitaal in Parow, maar ook die bou van ander hospitale in die Kaapprovinsie. Hulle het veral aandag bestee aan probleme wat in verband staan met röntgenterapie, isotope, mediese laboratoriums, metaboliese werk, en sentrale dienste soos sterilisering, die hou van rekords, ens.

WORLD LIST OF FUTURE INTERNATIONAL MEETINGS

ALTERATIONS AND ADDITIONS NOTIFIED DURING JUNE 1960

Third International Congress of Physical Medicine, Washington, D.C., 21 - 26 August 1960. Dorothea C. Augustin, Executive Secretary of the Congress, 30 N. Michigan Av., Chicago 2, Ill., USA.

Symposium on Initial Effects of Radiation on Living Cells, Moscow, 29 August - 2 September 1960. Prof. N. M. Sissakian, Deputy Scientific Secretary, Academy of Sciences of the USSR, Lenin Prospekt, Moscow, USSR. (UNESCO).

Fourth International Seminar of Medical Students, Budapest, September 1960. International Union of Students, Vocelova 3, Prague, Czechoslovakia.

Latin Medical Union, International Congress, Paris, 4 - 8 October 1960.

Bockus Alumni International Society of Gastroenterology, 2nd Biennial Scientific Meeting, Rio de Janiero, 17 - 21 October 1960. Dr. Figueiredo Mendes, Av. Rio Branco 257-18, Rio de Janiero, Brazil.

International Federation of Surgical Colleges, meeting, London, 3rd week October 1960. K. Cassels, Royal College of Surgeons, 47 Lincoln's Inn Fields, London, W.C.2, England.

Second Conference of Latin American Schools of Medicine, Montevideo, 28 November - 2 December 1960. Dr. Washington Buño, Secretario del Comité Organizador, Facultad de Medicina de Montevideo, Universidad de la República, General Flores 2125, Montevideo, Uruguay.

United Nations Educational, Scientific and Cultural Organization, Meeting of Experts on Terminology Used in the Care and Breeding of Laboratory Animals, Paris, November 1960. Place de Fontenoy, Paris 7e, France.

Karolinska Institute, Jubilee Congress, Stockholm, 3 - 7 December 1960. Swedish Tourist Traffic Association, Klara V. Kyrkogata 3 A, Stockholm, Sweden. To be held in conjunction with other festivities on the occasion of the 150th Anniversary of the Karolinska Institute.

Regional Symposium on the Use of Radioisotopes in the Study of Endemic and Tropical Diseases, Bangkok, December 1960. International Atomic Energy Agency, 11 Kärtner Ring, Vienna 1, Austria. In cooperation with the World Health Organization.

NUWE PREPARATE EN TOESTELLE : NEW PREPARATIONS AND APPLIANCES

FLUITRAN TABLETS

Scherag (Pty.) Ltd. announce the introduction of Fluitran tablets, and supply the following information:

Fluitran is a diuretic antihypertensive agent with activity 10-20 times that of hydrochlorothiazide, and 100-200 times that of chlorothiazide. It causes more equivalent sodium and chloride excretions, has a higher sodium to potassium-excretion ratio than chlorothiazide or hydrochlorothiazide, with lessened bicarbonate excretion and pH change. Fluitran permits a liberal salt intake and often eliminates 'no salt' diets in hypertensive patients. Its action persists for approximately 24 hours with the onset of effect within 2 hours and peak activity within 6 hours. It has a remarkably high degree of safety — clinical evidence of electrolyte imbalance is absent or rare. There is no refractoriness or diminution of response with continuous usage.

Fluitran is recommended as a *diuretic* for use in oedema associated with congestive heart failure, the nephrotic syndrome, hepatic cirrhosis, and premenstrual tension; oedema and toxaemia of pregnancy; and drug-induced oedema. Therapy with Fluitran may also be of value in oedema associated with obesity when used in conjunction with appropriate dietary measures.

Dosage. Usual dosage is 2 or 4 mg. taken once daily after breakfast. For initial diuresis, these doses may be given twice daily since a greater 24-hour effect is frequently obtained with such a dosage schedule. The maximum single effective dose of Fluitran is 8 mg. For maximum 24-hour diuretic effect this dose may be given twice daily. For maintenance a single 2-mg. tablet once daily often may be adequate, while doses as low as 1 mg. (one-half of a 2-mg. tablet) occasionally prove satisfactory.

Fluitran is of value as an *antihypertensive* both as primary therapy in the management of hypertension with or without the presence of oedema and in combination with other antihypertensive agents such as ganglionic-blocking agents and Rauwolfia alkaloids. The efficacy of other antihypertensive agents is enhanced when Fluitran is administered concomitantly. The dosage of antihypertensive agents should be reduced by 50% when they are given to patients already undergoing therapy with Fluitran. A similar reduction in dosage should be made if Fluitran is added to these drugs when they are being administered as primary therapy. In normotensive individuals Fluitran does not cause hypotension.

Dosage. Usual dosage is 2 or 4 mg. once daily with initial therapy often requiring these doses to be administered twice daily. The 4-mg. dose given once or twice daily is usually the most effective dosage initially. Higher doses would ordinarily not be required, but may be used initially.

Precautions.

It should be kept in mind that Fluitran is an extremely potent agent requiring low milligram doses for therapeutic effectiveness and therefore should be used accordingly. Electrolyte depletion may occur with over-vigorous therapy, and maximum doses should not be used repeatedly or frequently. Clinical evidence of electrolyte imbalance has been absent or notably infrequent. However, if there is any reason to suspect electrolyte imbalance, repeated serum and urine electrolyte determinations should be made and appropriate measures instituted. The doctor should consult the Schering Corporation USA Statement of Directions for details on indications, dosage, and administration precautions and contra-indications.

Packing. Fluitran tablets, 2 mg. and 4 mg. are supplied in bottles of 30 and 100.

Further information may be obtained from Scherag (Pty.) Ltd., P.O. Box 7539, Johannesburg.

BOEKBESPREKINGS : BOOK REVIEWS

MENSLIKE FISIOLOGIE

Menslike Fisiologie. Dele I en II. 3de uitgawe. Deur H. E. Brink, D.Sc.(Stell.). Pp. 606 en 477. Geïllustreerd. Deel I 57s. 6d. Deel II 42s. Stellenbosch/Grahamstad: Die Universiteits-Uitgewers en -Boekhandelaars. 1960.

In 'n tyd wanneer Afrikaans as taalmedium vir die onderrig van die wetenskap met rasse skrede vooruitgang maak, verteenwoordig hierdie twee boekdele oor *Menslike Fisiologie* 'n belangrike en tewens buitengewoon-gewigtige bydrae tot die Afrikaanse wetenskaplike literatuur. In die afgelope tyd het 'n aantal boeke oor die Anatomie en die Fisiologie, veral vir verpleegsters, in Afrikaans verskyn, maar sowel hul inhoud as hul terminologie het veel te wense gelaat. Brink se *Menslike Fisiologie* stel so 'n hoë standaard dat dit noodwendig 'n groot invloed moet hê, nie alleen op die skrywe van boeke oor die Fisiologie nie, maar ook op ander wetenskaplike werke in Afrikaans.

In die voorwoord bespreek die skrywer met duidelikheid en besadigdheid die netelige vraag van spelling en terminologie. Om heeltemal te verafrikaans of nie is die groot probleem, en professor Brink bied 'n kompromie aan wat, indien moontlik nog nie finaal nie, die meeste mense tevrede sal stel. In ieder geval, gedrogte soos ‚borsbeensleutel-beentepelvormige-slaapbeenuitsteekselspier' vir die sternomastoïedspier (wat 'n boek oor die Anatomie en die Fisiologie in ongenade bring en wat jare gelede gepubliseer is) is eens en vir altyd deeglik begrawe.

'n Skrywer van 'n handboek oor die Fisiologie word dadelik voor twee probleme gestel waaroor hy tot 'n gebalanseerde vergelyk moet kom: Eerstens, hoeveel biochemie en, tweedens, hoeveel kliniese materiaal moet vervat word. Op albei vrae voel die resensente dat professor Brink se boek te kort skiet aan 'n gebalanseerde inhoud. Met verwysing na die biochemiese aspekte van die onderwerp, word 'n aantal belangrike resente ontwikkelings oor die hoof gesien; onder andere, die sentrale rol van die sitroensuursiklus in metabolisme, die funksies van mitochondrië, nukleïensure, die biosintese van steroïedhormoon, hemoglobien A en hemoglobien S, die struktuur van insulien, oksitosien en vasopressien, en die metabolisme van cholesterol.

Die ouer en misleidende definisie van sure en basisse behoort vervang te word deur die meer moderne definisie ooreenkomstig die Bronsted-Lowry konsep. Die Henderson-Hasselbalch vergelyking wat Homer Smith beskryf as ‚een van die beroemdste matematiese vergelykings in die Fisiologie en die medisyne', word nie genoem nie. In die verslag oor die oksidasie van vetsure word slegs in die verbygaan melding gemaak van asetiel-koënsiem A. 'n Meer vrylike gebruik van chemiese formules sal die teks ophelder, byvoorbeeld, in die afdelings wat handel oor die biosintese van tiroïedhormoon, die ornitiensiklus, en ovariumhormoon.

Daar is deurgaans 'n neiging om die kliniese toepassings van die onderwerp te veel te beklemtoon. Die verstandigheid om die aandag van die student in die Fisiologie af te trek met omstandige verslae van tekens en simptome van siektes, kan sterk betwyfel word.

Die boek word oorvloedig geïllustreer en daar is baie diagrammatiese voorstellings. Nogtans, diagramme wat die anatomiese verwantskap van sekere organe aandui, kan geriefliheidshalwe uitgelaat word in 'n boek wat hoofsaaklik vir mediese studente geskryf is. Ernstige foute kom voor in 'n aantal diagramme wat metaboliese paaie aandui.

Die boek is besonder goed geskryf. Die styl is eenvoudig, direk en helder. Professor Brink moet gelukgewens word met 'n pionierswerk wat ongetwyfeld 'n klassieke werk in Afrikaanse wetenskaplike literatuur sal word. H.Z. A.v.Z.

TREATMENT OF URINARY LITHIASIS

Treatment of Urinary Lithiasis. Compiled and edited by Arthur J. Butt, B.S., M.D., F.A.C.S. Pp. xxii + 577. Illustrations. £8 8s. 0d. Oxford: Blackwell Scientific Publications Ltd. 1960.

This extensive symposium by 32 distinguished authors deals in its 512 pages with every aspect of urinary lithiasis.

Among its chapters are a historical review, a section on

anatomy, articles on the surgical and manipulative treatment of calculi in various parts of the urinary tract — even to the terminus of preputial calculi — and numerous outstanding contributions on medical management and pre- and post-operative care.

Colloidal chemistry, the rôle of infection, hyperparathyroidism, nephrocalcinosis, drug treatment, and each individual type of stone are dealt with in separate chapters.

The special interest of Dees in coagulum pyelolithotomy, of Hutch in the uretero-pelvic junction, and of Suby in the dissolution of calculi provide more recherché fare, and Mulvaney speculates upon the future of ultrasonic treatment.

There are a few minor criticisms. The author's deliberate simplification of the treatment of renal stone, for the sake of students, residents, and beginners, has led him to give rather too few details in this chapter. It is a pity, also, that partial nephrectomy is not considered and that the approach of Elmer Hess through the bed of the last rib is not described. Few urologists would whole-heartedly agree with the statement on page 426 that 'no stricture should be cut, through which any type of instrument may be passed'.

There are a few misprints which will doubtless be eradicated in succeeding editions which will certainly be called for.

The volume is beautifully, even lavishly produced. No urologist should fail to secure it, or, having secured it, will find it possible to abandon it unread. J.A.C.

BRIEWERUBRIEK : CORRESPONDENCE

INCOME LIMITS FOR MEDICAL AID SOCIETIES

To the Editor: I cannot believe that the rules governing the income limits for members of medical aid societies are being applied. Looking at them critically, I doubt if they could be applied. The expression 'gross income' needs definition, and the percentage rules seem quite ridiculous. These percentage rules appear to have been copied from the regulations governing sick benefit funds — a very different problem.

I find it strange that I have never met nor have heard of a medical aid society member whose benefits have been terminated because of his financial position; and I challenge any society secretary to prove to me that the income rules are being observed in letter or in spirit. This is not a small matter as some assert; it could undermine the whole fee structure of private practice.

Here in Durban the whole of industry and commerce will soon be covered by group schemes, which are offering cheap medical attention bar none, and which make great play with the statement that the lower fees are approved by our Association. How can I justify a fee of 1gn., when my own Association openly states that 12s. 6d. is a reasonable fee for a general practitioner consultation? If the patient is in the same income group, one or the other fee must be wrong.

How did the impression arise that the societies guarantee prompt payment in full? Item 2 of the General Preamble to the Tariff of Fees for Approved Medical Aid Societies says that such payment will only be made in so far as the society's rules and regulations allow. This is no guarantee at all, for if words mean anything, this proviso allows the society to veto the claim almost at will.

It is characteristic of the whole matter that Item 5 of the General Preamble instructs the practitioner to look at the patient's membership card, where any excluded benefits will be listed. I have looked to see, and the joke is on us. They are not there!

I know of wealthy members of group schemes who have continued to pay private fees rather than to let their doctor suffer from the fact that they have recently been forced to join such schemes. But in such circumstances Item 2 of the Preamble quite firmly lays the responsibility for full payment on the patient's shoulders. '*Any* responsibility of the medical aid society for payment of fees ceases' (my italics). The member has thus lost those benefits to which he was otherwise entitled, and for which his subscriptions qualify him. Is this loss fair?

It is analogous to the situation in the UK, established by an ideological government, whereby a patient who employs a doctor privately loses his right to the supply of drugs, which he could otherwise obtain freely through the State medical service. This injustice in Britain is now to be rectified, but we allow a similar state of affairs to continue, without protest.

As for 'prompt payment', the societies, according to the last paragraph of Item 2, will not even agree to move in the matter of an unpaid account, until 4* consecutive monthly accounts have been rendered. This appears to me to be granting at least 100 days credit. In no circumstances could this be considered a reasonable implementation of the above promise.

Finally, have our Federal Councillors read Item 7 of the Preamble carefully? It is most vicious. I am instructed by this Item, that if I once treat a medical aid society patient at tariff rates, I must for the rest of my professional life treat all medical aid society members at such rates, irrespective of who or what they are. It is surely repugnant to our Constitution, which expressly forbids our Association to act as a trade union.

I would welcome the news that these criticisms are unfounded, for many of my fellow general practitioners are, like me, most unhappy about the general trend of medical aid work. We believe it could be made to work to the benefit of us all, doctors and patients, but this will require a great deal more administrative care than is now being given to the matter.

N. R. Pooler

1201 Provident Assurance House
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18 July 1960

[*The last paragraph of Item 2 of the General Preamble to the Tariff reads. 'If payment of an account is not received after two consecutive monthly accounts have been rendered to the member, the third monthly account, giving the full name and address (home and business if possible) of the member, shall be sent directly to the Society concerned, bearing the words, written prominently in red: "Two months overdue, please investigate". The Medical Aid Societies have undertaken to expedite payment in these cases.' — *Editor.*]

SECOND INTERNATIONAL CONGRESS OF NEUROLOGICAL SURGERY

The Acting Secretary for Health of the Union of South Africa has kindly forwarded to the Association a copy of a letter addressed to the South African Ambassador in Washington by the Secretary-General of the World Federation of Neurosurgical Societies. This letter is reproduced below:

'The Second International Congress of Neurological Surgery, sponsored by the World Federation of Neurosurgical Societies, will be held in Washington, D.C., October 14 to 20 1961. The officers and committees of the Congress are developing a stimulating program which will include the presentation of important subjects in the field of neurosurgery by distinguished authorities. The program is not yet complete but will include four symposia on the following topics:

'I. Radioactivity and heavy radiation particles in neurosurgery.
'II. Space-occupying intracranial lesions.
'III. Re-evaluation of surgery in the treatment of pain.
'IV. A. Biology of the nervous system;
B. Hydrocephalus.

'The neurosurgeons of the United States and Canada, hosts to their foreign colleagues, invite all those engaged in this special field to attend. We hope there will be world-wide representation including neurosurgeons from South Africa.

'We shall be glad to keep you informed of further plans for the Congress as they develop and shall let you know the names of those from South Africa who will attend.

Bronson R. Ray
Secretary General

World Federation of Neurosurgical Societies
Office of the Secretary-General
525 East 68th Street
New York 21, New York.'